DIAPHRAGMATIC RESPIRATION: PSYCHOPHYSIOLOGICAL AND SUBJECTIVE EFFECTS IN AN ANXIOUS POPULATION

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By

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ABSTRACT

The present study was suggested by evidence in the psychological literature relating the presence of anxiety to rapid, shallow respiration. A three session breathing therapy program was compared to an attention control program on a number of self-report and psychophysiological variables. The research design was a pretest-posttest control group design. Subjects consisted of 18 self-referred, highly anxious adults, matched for sex and age, and randomly assigned to one of the two conditions. Both groups met for three, hour-long, small group (n=3) sessions. The breathing therapy program consisted of teaching slow, abdominal respiration as a relaxation technique and coping device. The attention control condition consisted of group discussion and self-relaxation. All subjects underwent two laboratory sessions, four weeks apart, during which respiration, cardiac, and skin conductance measures were obtained. Both tonic physiological activity over three periods (baseline, anticipation, and recovery), and specific responses to a stressor (loud noise) were assessed. Analyses were undertaken to determine between group differences on all measures. Repeated measures analyses of variance revealed breathing therapy to be significantly more effective than the attention control program in decreasing; (a) respiration rate, and (b) two self-report measures of anxiety. Limitations, implications, and contributions of this study are discussed.

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Relationships between mental states and respiration have been noted for a long time, back to the ancient belief that the diaphragm was the center of the soul. In the scientific literature many studies are reported relating respiratory patterns with variables such as personality (Alexander & Saul, 1940; Skarbek, 1970); problem-solving ability (Golla & Antonovitch, 1929), life situations (Jacobs, Spilken, Norman, & Anderson, 1970), and emotions (Cohen, Goodenough, Witkin, Oltman, Gould, & Schulman, 1975; Dudley, Holmes, Martin, & Ripley, 1963; Feleky, 1916; Roessler, Bruch, Thum, & Collins, 1975; Stevenson & Ripley, 1952). Probably the best established and most often studied relationship is between anxiety and respiration. That such a connection exists is supported by many lines of evidence from chemical studies to case reports. The evidence, discussed below, strongly supports the relationship between rapid, shallow, irregular breathing and anxiety.

Despite support for this relationship, there is inconclusive evidence that by consciously altering one's pattern of respiration (i.e., breathing slowly, deeply, and regularly) one can effect a decrease in anxiety. Although information from several sources suggests this may be a most useful technique in decreasing tension, carefully controlled studies are necessary for validation of these claims.

Respiration and Anxiety

Breathing Patterns. Somatic arousal (increased heart rate, muscular tension, faster respiration, etc.) is concomitant with subjective feelings of anxiety. This arousal is usually considered a manifestation of an underlying anxiety state. Specifically, high anxiety has consistently been associated with rapid and shallow respiration (Dalessio, 1978; Damas Mora, Grant, Kenyon, Patel, & Jenner, 1976; Faulkner, 1941; Martin, 1961; Suess, Alexander, Smith, Sweeney, & Marion, 1980). As well, many studies have demonstrated that respiratory patterns of people labeled "anxiety neurotics" are consistently different from those of "normals" (Christie, 1935; Clausen, 1951; Feleky, 1916; Malmo & Shagass, 1949; Sutherland, Wolf, & Kennedy, 1938).

Using a recording spirometer, Christie (1935) found that those patients with a clinical diagnosis of anxiety neurosis showed a shallow, irregular type of respiration. However, no control groups were described, and this pattern of respiration was also found in advanced emphysema.

Utilizing a pneumographic recording technique in groups of normal and neurotic subjects, Clausen (1951) recorded thoracic and abdominal respiratory movements on five subsequent days. Measures subjected to analysis consisted of respiration rate and inhalation/exhalation ratios for both thorax and abdomen. It was found that neurotics breathed more rapidly and showed more irregularity within sessions than normals.

Sutherland et al. (1938) obtained spirometric records from hundreds of clinically diagnosed neurotic and psychotic patients which differed from those of normals. The abdominal records are characterized by irregularities in respiration and by periodic gasps and sighs. They call these recordings "fingerprints" of mental states because recordings taken three to twenty-eight days apart duplicated each other with great precision, and as clinical status improved, respiratory patterns tended to approach normality.

Malmo and Shagass (1949) found respiratory response to painful stimuli to be much greater, as determined by the amount of deviation from prestimulus respiration pattern (irregularity scores), in anxious patients than in either normals or schizophrenics.

Lum (1975, personal communication) reports having done impedance spirometric analyses and blood carbon dioxide levels on more than 2000 patients suffering from anxiety states. Impedance spirometry allows one to measure various aspects of respiration such as rate, rhythm, and thoracic movement by recording volume changes between pairs of electrodes applied to the chest wall. Results have shown an abnormal breathing pattern to be universal in these cases, characterized by predominant use of the thorax, little diaphragmatic movement, irregular volume, and frequent sighs. An abnormally low level of carbon dioxide is also reported to occur in the majority of thoracic breathers. Unfortunately, criteria for the diagnosis of "anxiety states" are not specified clearly. Thus results may only be applicable to those suffering from aberrant breathing in the first place.

These studies support the contention that somatic arousal as reflected by respiration is an important characteristic of anxiety states. In order to more fully appreciate the relationship between anxiety and respiration however, it is necessary to understand the mechanics of breathing, and how various parameters of respiration effect chemical changes associated with somatic arousal.

<u>Mechanics of Breathing</u>. Internal respiration is a complex chemical process in which oxygen is carried by the blood to nourish every cell in the body. The efficiency of this process depends in considerable part on the mechanics of air intake, or external respiration (Stein & Luparello, 1967).

External respiration refers to the process by which oxygen is obtained from the atmosphere, through the nose, and down into the lungs. This mechanical process depends on expansion of the torso to draw in air. The torso may be expanded in three ways; lifting the shoulders, extending the chest outwards, or expanding the diaphragm downwards. These three types of breathing may be termed clavicular, thoracic, and diaphragmatic. Although upon maximal exertion all three types of breathing are utilized, the most efficient for normal functioning is diaphragmatic breathing (Brena, 1973; Rama, Ballentine, & Hymes, 1979). This is because diaphragmatic breathing pulls oxygen to the lower portion of the lungs where most of the blood necessary for oxygen uptake circulates.

Thoracic breathing is much more shallow, allowing air only into the middle and upper portions of the lungs, and as such is less efficient as an habitual mode of respiration (Brena, 1973). It takes considerably more effort to obtain sufficient oxygen using primarily the thoracic mechanism, and since more work is required, more oxygen is needed. This results in an increased respiration rate, and may contribute to feelings of fatigue and breathlessness, especially upon exertion (Rama et al., 1979; Soley & Shock, 1938).

There is some evidence that thoracic or shallow, rapid respiration is more often related to anxiety than is diaphragmatic breathing, both in "anxiety neurotics" and in normal people facing threatening situations (Clark, 1978; Clarke & Hemsley, 1982; Cohen et al., 1975; Harris, Katkin, Lick, & Habberfield, 1976; Holmes, McCaul, & Solomon, 1978; McCaul, Solomon, & Holmes, 1979; Scopp, 1975). Also, Damas Mora et al. (1976) and Martin (1961) demonstrated a linear relationship between state anxiety and respiration rate. Thoracic breathing is typically activated as a biologically adaptive response to stress or threat, providing the extra oxygen necessary to either fight or flee. If no activity occurs to discharge energy, a vicious circle may ensue in which this overbreathing exacerbates and maintains feelings of tension (Lewis, 1959). If this reaction is intense enough or occurs frequently, this type of breathing may become habitual. It has been repeatedly demonstrated that this overbreathing response is very easily conditioned and generalization often occurs to a wide variety of environmental stimuli (Bykov, 1959; Freedman, 1951).

Hyperventilation Syndrome. Lum (1976), Salinsky (1974), and others (Kerr, Dalton, & Gliebe, 1937; Lewis, 1959; Rice, 1950) convincingly argue that overbreathing itself may be a primary disorder which can result in symptoms generally associated with, and often labeled, anxiety neurosis. Some people adopt habitual modes of breathing which may be harmful. Habitual overbreathing, characterized by rapid, thoracic respiration and forced exhalation (i.e. frequent sighs) is a common breathing disorder which may have profound effects on many physiological systems.

The respiratory system regulates ventilation to maintain arterial blood tension levels of carbon dioxide (CO2) and oxygen (O2) at an optimal point, meeting the needs of the body for O2 as well as for CO2 removal and regulation of blood acidity (Comroe, 1974). The most immediate effect of overbreathing, especially forced exhalation, is a drastic reduction of CO2 in the blood, producing respiratory alkalosis (increased alkalinity of the blood). In most individuals, emotional arousal is initially correlated with reduced CO2 levels (Dudley, Martin, & Holmes, 1964). This mild hyperventilation occurs when ventilation of the lungs is greater than that needed to eliminate CO2 (Comroe, 1974). Carbon dioxide therapy (breathing into a paper bag, for example) works in part to decrease anxiety by replenishing excessive loss of CO2 (Aveni & Cutter, 1977; Halsam, 1974; Wolpe, 1958). Loss of CO2 in turn, decreases the availability of calcium, leading to enhanced nerve excitability manifested in muscle tremors and spasms. Pitts and McClure (1967) have been intrigued by the similarity between many symptoms of anxiety neurosis and those seen in conditions where calcium ions are low. They provide data suggesting that decreased levels of calcium ions, associated with respiratory alkalosis in the hyperventilation syndrome, may contribute to anxiety states (also Halsam, 1974).

Noticeable symptoms frequently resulting from the above physiological changes include heart palpitations, dizziness, shortness of breath, epigastric pain, muscular tremors, anxiety, and weakness. In fact, Lum (1981) states that almost any organ or system may be affected. This is hardly surprising when one considers that the function of breathing itself is to nourish every cell of the body.

Lehrer and Woolfolk (1982) report on a new anxiety inventory assessing somatic, cognitive, and behavioral aspects of anxiety. The somatic factor consists of a variety of hyperventilation symptoms. Interestingly, in various validation studies using both normal and anxious subjects, it was found that the somatic factor correlated very highly with many well known anxiety scales such as Spielberger's Trait anxiety (Spielberger, Gorsuch, & Luschene, 1970), Eysenck's Neuroticism Scale (Eysenck & Eysenck, 1968), phobic and general anxiety scales of the SCL-90 (Derogatis, 1977) and the IPAT anxiety inventory (Krug, Scheier, & Cattell, 1976). These results offer further support for the relationship between hyperventilation and anxiety states as measured by commonly used clinical questionnaires.

While it is fairly well established that habitual overbreathing is often associated with anxiety states, it is less clear whether the association is a causal one. Although physiological studies support the relationship between low levels of arterial carbon dioxide and various somatic and psychological symptoms, the findings are hardly definitive and often inadequately reported. Further support for the theoretical background of the hyperventilation syndrome should come from carefully controlled experimental studies rather than educated speculation.

Therapeutic Value of Controlled Breathing

The studies reported above suggest that respiratory irregularity as reflected by increased rate, variable depth, and arrhythmicity accompany, or perhaps cause, subjectively uncomfortable states in both normal and anxious populations. Thus it would appear that by eliminating these irregularities, via teaching proper methods of breathing, one could effect a decrease in anxiety. There have been many claims made concerning the efficacy of correct breathing behavior in decreasing anxiety and tension (Geba, 1973; Haugen, Dixon, & Dickel, 1960; James, 1924; Lum, 1976, 1981, personal communication; Proskauer, 1968; Schade, Hruza, Washburne, & Carns, 1952). For the most part these claims are scientifically unsupported, although clinical results are often impressive. Lum (personal communication), for example, proposes a treatment of breathing awareness and teaching a regular, slow, diaphragmatic pattern of respiration. Using these techniques on over 2000 patients suffering from "anxiety states", Lum reports an 80% cure rate defined as "no symptoms, no anxiety, and no drugs required". Fifteen per cent are relieved of the "vast majority of symptoms", and 5% are described as "quite intractable". No control groups were utilized however, as that would entail delaying treatment. Gibson (1978) suggests that the validity of Lum's breathing therapy program could be enhanced by applying it in conjunction with an informed background of behavioral principles.

The control of breathing is a common component of many anxiety reduction techniques such as Stress Innoculation Training (Meichenbaum, 1977), Progressive Muscle Relaxation (Bernstein & Borkovec, 1973), Conditioned Relaxation (Paul & Berstein, 1973), the Relaxation Response (Benson, 1974), and various meditation techniques. It is not clear how much of the relaxation achieved by these methods is due to the controlled breathing component or to other aspects of the treatment procedure (Lader & Marks, 1971). The inclusion of controlled breathing in all of these effective relaxation techniques suggests that this alone may be effective in reducing anxiety.

It has been found that other physiological systems such as heart rate (Deane, 1964, 1965; Holmes, Solomon, & Buchsbaum, 1979; Westcott & Huttenlocher, 1961; Wood & Obrist, 1964) and skin potential (Harris et al., 1976; McCaul et al., 1979) vary with rate and depth of respiration. Respiration is also the only physiological system associated with autonomic arousal that is easily brought under voluntary control.

Therefore, if respiration is controlled during bouts of anxiety there will be fewer physiological cues to maintain and enhance the anxiety reaction. As a self-control technique, controlled respiration is easy to learn, and since it is "portable", may be applied in any situation.

<u>Scientific Studies of Controlled Breathing</u>

In spite of strong evidence supporting a relationship between respiration and anxiety, and impressive clinical support for the benefits of controlled respiration, very few experimental studies have investigated the use of this procedure to decrease anxiety. The studies which have been conducted may be divided into two types: controlled respiration as (a) a technique for controlling stress induced in laboratory situations and (b) a method of anxiety reduction.

In the first category, Harris et al., (1976) evaluated the effects of paced respiration (8 cycles per minute) in modifying autonomic responses to stress in normal subjects. Comparing a paced respiration group to attention and baseline control conditions, they found that subjects in the paced respiration group showed less autonomic responsiveness to an electric shock and its anticipation. The results for electrodermal measures indicated that pacing respiration can reduce autonomic responsivity to a stressful stimulus.

Holmes et al., (1978) required subjects to either (a) breathe in such a way as to replicate breathing patterns obtained during a rest period, (b) turn a knob to replicate resting breathing patterns (attention control) or (c) sit quietly. After the tracing manipulation was introduced, one-half of the subjects in each group were threatened with a series of shocks. It was predicted that the respiratory tracing condition would show less arousal under threat conditions. Results did not support this hypothesis as the no-tracing condition was the only one to show a decrease in arousal over time. However, in this study, rate of respiration was not specifically manipulated, and it may be a crucial variable.

Clark (1978) examined the effects of paced respiration on autonomic and self-report measures of subjects with high dental anxiety during exposure to films of dental surgery. Subjects were trained (in 10 minutes) to breathe at either 8, 16 or 24 cycles per minute or were assigned to attention or no treatment control conditions. The pacing procedure failed to induce any meaningful physiological changes as measured by electrodermal response and heart rate. The lack of electrodermal change may have been due to the non-threatening nature of the initial film segments which possibly enhanced habituation to the later, more threatening scenes (Clark, 1978). The lack of change in heart rate may have been due to manipulating only rate, not depth of respiration. This supports the conclusions of Sroufe (1971), and Wood and Obrist (1964), that respiration depth rather than rate exerts a primary effect on heart rate levels. However, potent effects on self-report measures were found, with the slow respiration group reporting the least discomfort and the rapid respiration group reporting the greatest discomfort. This study suggests that subjective appraisals of distress may be effectively altered by respiration pacing, independent of visceral changes.

McCaul et al., (1979) investigated the efficacy of three conditions; (a) breathing regulated at one-half normal rate, (b) breathing regulated at normal rate, and (c) breathing not regulated, in reducing arousal to threat of shock. Expectancy was also controlled by informing one-half of the subjects in each group that the procedure would help them to relax. Results indicated that slowing respiration rate reduced physiological arousal as measured by skin resistance and finger pulse volume, and reduced self-reports of anxiety. Expectations did not influence arousal. These data provide evidence for the effectiveness of paced respiration as a coping strategy.

In the second category, three studies were found in the literature which dealt with controlled breathing as a therapeutic technique for anxiety reduction.

Datey, Dehmukl, Dalvi, and Vinekar (1969) demonstrated a diaphragmatic breathing technique, known as Shavasan, to be instrumental in improving the condition of hypertensive patients as indicated by lower blood pressures, less use of medication, and subjectively reported improvement. They report the essential requirements to be use of the correct technique, regularity in performing the exercise, and a quiet environment. Unfortunately, no control groups were employed in this study.

Scopp (1975) compared four groups; progressive muscle relaxation, breathing treatment, combination of both muscle relaxation and breathing, and an attention control on a variety of cognitive and affective measures including Spielberger et al.'s (1970) State Trait Anxiety Inventory (STAI) and various measures of intelligence. Results indicated that all three treatment groups significantly decreased in state and trait anxiety. Heart rate, respiration rate, and skin conductance were monitored for six volunteers in the full treatment and control groups at pre and posttreatment. Although no statistical analyses were performed on these data, it is reported that subjects in the full treatment group showed an overall change in the expected direction on these measures whereas no change was noted in the attention control subjects. Unfortunately, physiological changes were not assessed for the separate breathing and muscle relaxation groups, and only a very few of the total sample (N = 260) were assessed. Scopp concluded that deep, slow breathing be used in conjunction with muscle relaxation to maximize reduction of state anxiety in individuals.

Foss (1975) tested the effectiveness of a three hour Breathing Therapy program for reducing anxiety in self-referred college students. Dependent measures consisted of the STAI and the Wolpe and Lang (1964) Fear Survey Schedule (FSS). Results showed that the Breathing Therapy program was significantly more effective in decreasing both state and trait anxiety than a no treatment control. Unfortunately, an attention control group was not included to rule out expectancy effects, and no assessment of physiological changes was carried out.

Although relatively few experimental studies have been done, those that have been reported suggest controlled breathing to be effective in decreasing anxiety. These findings lend support to the claims made by some clinicians concerning controlled breathing. However results have been variable. The studies differ on a number of important variables such as how breathing is controlled, type of subjects, and especially outcome measures. Further research in this area should attempt not only to replicate some of the significant findings described above, but also to investigate which measures are sensitive to changes in respiration patterns. It is important that each of the three systems of anxiety; behavioral, cognitive, and physiological be assessed. Due to both the paucity and variability of the data thus far, it is clear that more research is needed in this area.

Problem

Before complex comparison studies are undertaken, there should be clearer evidence that, relative to attention control conditions, a breathing therapy treatment is effective in decreasing anxiety. Ideally effects should be demonstrated in more than one of the three systems of anxiety; physiological, cognitive, and behavioral. However, to date, the studies of breathing therapy have focused primarily on self-report measures of anxiety. Studies using paced respiration to decrease somatic arousal in normal subjects, as well as the physiological literature on breathing, suggest that respiratory control may be effective in reducing physiological arousal as well.

Effects should also be demonstrated for a group that is initially high in anxiety. Lehrer (1978), and Lehrer, Shoiket, Carrington, and Woolfolk (1980) have demonstrated that physiological reductions of anxiety are more prominent in patients with a clinical diagnosis of anxiety neurosis than in normal subjects. Using high anxiety subjects also provides a better picture of the clinical utility of a technique, and Scopp (1975) reports that high anxiety is a good predictor of benefit derived from various relaxation training procedures.

In view of the above, it would be interesting to further determine if controlled respiration is a useful technique for decreasing anxiety, both in general, and in response to a stressor.

Hypotheses

The primary hypothesis of this study is that teaching deep, slow, rhythmic respiration is more effective than an attention control condition in reducing self-reported anxiety, and physiological activity during a stressful situation, in an anxious population.

It is also hypothesized that anticipation of a loud, high-frequency tone will act as an effective stressor, increasing physiological activity over baseline levels.

METHOD

Subjects

Subjects were solicited by means of an advertisement for a research/treatment program placed in the local newspaper. Eighteen subjects between the ages of twenty-five and fifty-five, consisting of eight males and ten females completed the entire program. All subjects were high in trait anxiety as indicated by a score of 34 (77th percentile) or higher on the IPAT Anxiety Scale Questionnaire (ASQ). As well, eleven of the subjects had undergone psychiatric treatment in the past for anxiety-related problems, indicating an approximate clinical population. Other inclusion criteria consisted of willingness to sign a participation contract (to minimize attrition), no organic respiratory disorders, no current use of formal relaxation techniques such as progressive muscle relaxation, transcendental meditation or yoga, no use of major tranquilizers in the past six months, and no regular use of minor tranquilizers.

Measures

Paper and Pencil Measures. The IPAT Anxiety Scale Questionnaire (Krug et al., 1976) is a brief scale developed as a means of getting clinical anxiety information in a rapid, objective, and standard manner (Krug et al., 1976). It is commonly used in research as a screening device for subject selection and, as norms are available for the general adult population, it is ideal for this purpose. Test-retest and internal reliability are high. Validity has been assessed several ways and it reportedly correlates highly with other measures of anxiety (Krug et al., 1976). The IPAT ASQ has also been shown to be sensitive to changes in anxiety level due to therapeutic intervention (Ferguson & Gowan, 1974; Sherman & Plummer, 1973; Wilson & Wilson, 1970).

The <u>State Anxiety Scale</u> (A-State) of the State-Trait Anxiety Inventory (Spielberger et al., 1970) consists of 20 statements, each rated on a four point scale indicating how a subject feels at a particular moment in time. The essential qualities evaluated by the A-state scale involve feelings of tension, nervousness, worry, and apprehension. It may be used to determine the actual levels of A-state intensity at a given moment, or with reference to some identified event or situation. This scale may be administered quickly, is easily understood, and is sensitive to changes over time (Spielberger et al., 1970).

The <u>Fear Questionnaire</u> (Marks & Mathews, 1979) is a one-page, self-rating form designed to monitor change due to intervention in phobic patients. The form yields four scores: main phobia, global phobia, total phobia, and anxiety-depression. The degree of avoidance of these phobic situations is assesed on a scale from 0 (would avoid it) to 8 (would always avoid it). This rating gives an indication of behavioral response to these events. Items making up the total phobia and anxiety-depression scales were included on the basis of high factor loadings on agoraphobic, social, tissue damage, and anxiety-depression factors derived by three independent factor analytic studies (cf Marks & Mathews, 1979). Test-retest reliabilities for all items are high, from .79 to .96, based on a retest interval of one week with 20 phobic patients (Marks & Mathews, 1979). There is also evidence of external validity in that the total phobia and anxiety-depression scores reflect the clinical status of the patient (Ginsberg & Marks, 1977; Marks, Hallam, Philpott, & Connolly, 1977). The questionnaire has also been shown to be sensitive to clinical improvement after treatment in a sample of 26 phobic patients (Marks & Mathews, 1979).

The Anxiety Scale (Hart, unpublished, 1983) consists of three lists of 8 or 9 adjectives representing a continuum from "very anxious" to "feeling great" (see Appendix A). The subject chooses one adjective on each list describing how he/she has been feeling during the past few minutes, or how he/she is feeling at the present moment. Each adjective is weighted differently and the score consists of the average weight from all three lists. The scale values were established by having 58 students assign ratings of -5, to +5 to a list of 50 adjectives considered to be indicative of relaxation, confidence, and tension/anxiety. Words for the scales were selected to (a) provide a distributed range of mean values, and to (b) have small standard deviations (evidence of consensus rating). Three psychology classes (total N=127) were administered the scale on two occasions each, once at the beginning of a regular lecture and once prior to a mid-term quiz. Cronbach alphas for the two occasions were .79 and .87. The scale total score clearly discriminated between the two conditions ($t=13.5$, $p<.0001$) which are presumed to differ in level of stressfulness, thus providing evidence of the validity of the scale.

The Somatic Inventory was taken from a questionnaire recently developed by Lehrer and Woolfolk (1982) to assess somatic, cognitive, and behavioral aspects of anxiety (see Appendix B). Each item is rated on a 9 point Likert type scale according to its frequency of occurrence, one indicating "never", nine indicating "almost always". The items were drawn from the Minnesota Multiphasic Personality Inventory, the State-Trait Anxiety Inventory (Spielberger et al., 1970), and the authors' own clinical experiences (Lehrer & Woolfolk, 1982). All items on the somatic scale of the inventory were shown by factor analysis to have a high loading on a somatic factor. According to Lehrer and Woolfolk (1982), most of the items on the somatic scale appear to describe the symptoms produced by hyperventilation. Split-half reliability of the somatic scale was shown to be .85 (Lehrer & Woolfolk, 1982). Some discriminant and convergent validity for the somatic scale were obtained from the SCL-90-R (Derogatis, 1977). The SCL-90-R somatization scale was found to be closely related to the somatic scale, but the later was not found to correlate highly with the SCL-90-R psychoticism scale. There are also significant relationships between the somatic scale and the IPAT anxiety inventory (Krug et al., 1976), the Eysenck Personality Inventory neuroticism scale (Eysenck & Eysenck, 1968), and the Hamilton Rating Scales (Hamilton, 1959).

Unfortunately no normative data are yet available for the Lehrer and Woolfolk trimodal inventory, but the somatic scale may be a useful pre-posttreatment indicator of change in somatic anxiety.

The Anxiety Diary (Appendix C) provides a self-report index of behavioral reactions to stressful situations. Subjects indicate each day at least one anxiety-evoking situation, their reaction to it, and method of coping. Also, subjects rate on a scale of one to ten how well they felt they dealt with the specified situation, one indicating they felt they used a very poor coping strategy, ten indicating a very good strategy was employed.

The Credibility Questionnaire, (Appendix D) adapted from Borkovec and Nau (1972), consists of three, ten-point scales assessing the credibility of therapy and placebo control conditions. Higher scores on this questionnaire indicate higher subject credibility. It is important for all conditions in a treatment study to be equally believable as otherwise results may be due to differences in expectancy.

The Postexperimental Questionnaire (Appendix E) is designed to elicit a subjective appraisal of the entire program. Subjects are asked their feelings concerning the program and the therapist, if the program met their expectations, and if they have any suggestions for improvement.

Stress Induction, Physiolgical Measures, and Apparatus. Respiration, heart rate, and skin conductance measures were recorded during laboratory sessions on a Beckman model R-411 dynograph. Instructions, warning tone, and noise stimulus (stressor) were recorded and administered with a Sony TC-252 stereo tape recorder. Subjects were seated upright in a comfortable chair. Most physiological measures were scored for nine, thirty-second epochs, consisting of the last ninety-seconds of baseline (epochs 1, 2 and 3), the ninety second anticipation period (epochs 4, 5 and 6), and a ninety second recovery period (epochs 7, 8 and 9).

The stress induction procedure described above is similar to that used by Lehrer (1978), Lehrer et al., (1980), and Shapiro and Lehrer (1980) in assessing physiological effects of various types of relaxation techniques. However, each of these studies used five aversive tones instead of one because habituation was also being studied. A stress procedure was deemed important to this study because Lehrer (1978) found that differences between relaxation trained and untrained subjects were revealed more clearly when subjects were tested under stressful conditions, as opposed to relaxing conditions. Although electric shock and its anticipation have been used in other studies (Harris et al., 1976; Holmes et el., 1978; McCaul et al., 1979) as an effective stress manipulation, it was felt that this would be too intrusive a procedure for use with anxious subjects, possibly causing high attrition. Minimal between-session habituation to the noise was expected to occur as it has been shown that those with high anxiety do not habituate easily to repeated presentation of noxious stimuli (Lader & Wing, 1966).

Thoracic and abdominal records of respiration patterns were obtained using two channels of the dynograph (with Beckman type 9853A couplers) as suggested by Grossman (1967). Beckman type 7001 respiration strain guage belts were placed on the subjects; one around the chest as close as possible to the armpits to record thoracic amplitude, and one placed half-way between the lowest point on the rib-bones and the highest point on the hip-bone to record abdominal amplitude (Svebak, 1975). Respiration rate was assessed by counting the number of complete cycles within each period and computing group averages for the three periods. Mean abdominal and thoracic depths in millimeters were sampled from the first three cycles of each epoch and group means calculated for each period.

Heart rate was measured through a Beckman type 9857 cardiotachometer, from Beckman biopotential electrodes attached to the right wrist and left ankle (lead II configuration) with a ground on the right ankle. Maximum and minimum heart rates were determined for each of the nine epochs using the peak rate method smoothed, recommended by Opton, Rankin, and Lazarus (1965) and the low rate method (Clifton & Graham, 1968), also smoothed. A smoothed score is obtained by averaging each data point with preceeding and succeeding data points. These measures were then averaged to obtain a mean peak and a mean low heart rate for each period. Using the peak and low rate methods eliminates most of the irrelevent variation due to sinus arrhythmia which is accentuated by imposition of respiration control procedures

(Harris et al., 1976; Opton et al., 1965; Westcott & Huttenlocher, 1961). For this reason, these measures are preferred to other commonly employed techniques such as averages or "peak to valley" scores (Lang & Hnatiow, 1962). In order to evaluate cardiac responses to the stressful stimulus, raw heart rate data (unsmoothed) were used. Heart rate acceleration and deceleration responses to the stimulus were determined relative to baseline (epochs 1, 2, and 3) maximum and minimum levels. Heart rate accelerations were calculated by subtracting the maximum heart rate during baseline from the maximum poststimulus heart rate within ten seconds of stimulus onset (Lehrer et al., 1980). Thus, a positive value for heart rate acceleration indicates a higher maximum heart rate after the stimulus, or an accelerative response. Heart rate decelerations were calculated by subtracting the minimum poststimulus heart rate within ten seconds of stimulus onset from the baseline minimum heart rate (Lehrer et al., 1980). A positive value for heart rate deceleration indicates a lower minimum heart rate after the stimulus, or a decelerative response. Group means of accelerative and decelerative responses were then subject to analysis.

Electrodermal activity was detected by Beckman biopotential (.6cm sq., Ag/AgCl) skin electrodes, using Beckman NaCl electrode paste as the contact medium. The electrodes were placed on the thenar and hypothenar eminences of the left palm. A type 9842 coupler with a constant voltage (.5 volts) circuit enabled direct measurement of skin conductance. Three measures of skin conductance responsivity were obtained. Mean frequency of spontaneous skin conductance responses (SSCRs) above .1 micro mho in amplitude was assessed during the last sixty seconds of each of the three periods. This was done to eliminate any specific responses due to instructions, warning tone and stimulus (Stern & Anschel, 1968). Amplitude of the skin conductance response (SCR) evoked by the stimulus (loud tone) was measured. The recovery rate of this evoked SCR was measured temporally by dividing the total number of seconds to recovery by two (SCR rec.t/2) (Venebles & Christie, 1980). Edelberg (1972) has found that stress slows recovery of SCRs. Amplitude measures are expressed in terms of log conductance as this transformation tends to normalize the distribution and minimize any correlation with initial level (Johnson & Lubin, 1972; Lykken & Venables, 1971). To avoid log transformations of zero (no response), one was added to all SCR amplitude scores before the data were transformed (Venables & Christie, 1980).

Subject Handouts

During the first treatment session, subjects in the experimental group were given a handout explaining the rationale for respiration control as a means of reducing anxiety (Appendix F). This handout, entitled Anxiety Management Training Manual, is based in part on literature about stress (Cannon, 1928; Wallis, 1983), and in part on the literature relating respiration to anxiety (Brena, 1973; Lum, 1975, 1976, 1981; Rama et al., 1979). This printed rationale ensured that each of the three experimental treatment groups received exactly the same information about breathing therapy, and provided a reference between sessions.

Experimental subjects were given another handout entitled Diaphragmatic Breathing Instructions, consisting of eleven instructions (Appendix G) taken from the literature on relaxation and breathing techniques (Benson & Klipper, 1975; Bernstein & Borkovec, 1973; Cooke, 1979; Datey et al., 1969; Gibson, 1978; Innocenti, 1966; Lum, 1975, 1976, 1981; Paul & Bernstein, 1973; Rama et al., 1979). This handout ensured uniform practice techniques among experimental subjects, and provided a reference between sessions.

Subjects in the control groups were given a handout during their first session, also entitled Anxiety Management Training Manual (Appendix H). This handout, given to enhance the credibility of the control program, contains information about anxiety exclusive of any specific relaxation instructions. Information for this manual was taken from sources about stress (Cannon, 1928; Wallis, 1983), and cognitive theory (Meichenbaum, 1977).

Subjects in both groups were given, and asked to sign a participation contract (Appendix I), specifying the terms of the research program, and designed to decrease attrition. Subjects were also asked to sign homework contracts, specifying the place and time of their relaxation sessions (Appendix J). Lastly, subjects were given homework completion charts (Appendix K) which they were to fill out each time they practiced their relaxation technique. This chart was brought to each therapy session and served as a basis for reinforcement from the therapist. Foss (1976) showed that subjects rated their experience with a breathing therapy program more highly, when required to do homework via the use of contracts and charts, than subjects without required homework. The trends also demonstrated more relaxation practice, and self-reports of the technique being more effective when homework was required (Foss, 1976).

Procedure

Initial contact with potential subjects was made by telephone. A screening interview was scheduled for all interested people. At this initial interview potential subjects were first administered the IPAT ASQ which was immediately scored to determine eligibility. Data were collected concerning subject characteristics; age, sex, health, medication, experience with relaxation techniques, and prior therapy (see Appendix L). Qualified subjects were then asked to sign a participation contract specifying compliance with all aspects of the program to the best of their ability, and agreement to deposit $10.00, refundable upon completion of the program (see Appendix I). An inventory of hyperventilation symptoms (from Lehrer & Woolfolk, 1982) was administered to obtain data on the relative frequency of these symptoms (see Appendix B). Subjects completed the Fear Questionnaire (Marks & Mathews, 1979), identifying at least one personal situation frequently encountered causing them to feel anxious. The State anxiety scale of the STAI was completed with reference to how they would expect to feel should that situation be encountered within the next few days. At the end of the initial interview each subject was scheduled for the first laboratory session.

Subjects participated individually in two laboratory sessions approximately four weeks apart, one before therapy and one afterwards. These laboratory sessions were introduced to subjects as "stress-tests". Subjects were asked to refrain from smoking for thirty minutes prior to psychophysiological assessment (Martin & Venables, 1980). The Anxiety Scale (prestress) was completed with reference to the impending stress-test. Skin conductance, heart rate, and respiration measures were recorded for baseline, anticipation, and recovery periods. Electrodes were attached for ten minutes prior to recording baseline data to allow acclimitization to the laboratory and to permit calibration of the polygraph. Subjects were instucted to get as comfortable as possible during this time. A tape-recorded message then revealed all further instructions. After baseline data were recorded for three minutes, subjects were informed that sometime after a warning tone, they would hear a very loud, high-pitched burst of noise. They were asked to try to remain relaxed. A tone then indicated the beginning of an anticipation period lasting ninety seconds. This period terminated with a 1000 Hz, 100 decibel burst of noise lasting 3.5 seconds from two speakers placed two feet behind each side of the subject's head.

Measures continued for a ninety second recovery period (see Appendix M for verbatim instructions). Subjects were then asked to rate on a scale from one (not at all) to ten (extremely) how distressing they found the noise to be. After the first stress-test subjects were informed that they would be contacted within two weeks concerning the dates and times of the three treatment sessions.

After all subjects had participated in the first physiological assessment, they were paired as closely as possible according to sex, age, and cigarette consumption. Subjects from each pair were then randomly assigned to either the treatment or control condition. This assured that both groups, although chosen at random, were of similar composition on these possibly crucial variables.

The treatment programs consisted of three, one-hour sessions of small group training in either the experimental or attention control condition. These sessions were from three to five days apart for each group. The posttreatment stress-test was identical to the above described procedure except the experimental group was instructed to relax using the breathing techniques learned during treatment (Appendix M).

During the posttreatment assessment session, psychophysiological recordings were conducted as described above. Subjects then completed the Credibility and Postexperimental Questionnaires (see Appendixes D & E), A-state scale of the STAI with reference to their personally identified, anxiety-arousing situation, the Fear

Questionnaire, and the IPAT ASQ. Subjects were refunded their $10.00 deposit.

Experimental and Control Conditions

The experimental program, designated as 'anxiety management training', consisted of three, one-hour sessions conducted in small groups (n = 3), from three to five days apart.

The sessions were scheduled to best accommodate the participants, and were held in the Memorial University Psychology Clinic on Elizabeth Avenue. Subjects completed the Anxiety Scale (postrelaxation) after the first and last relaxation sessions to assess the effectiveness of the breathing technique in inducing relaxation. A brief session by session description of the experimental program follows.

Session one. Subjects were given the handout explaining the rationale for respiration control in reducing anxiety. They were asked to read it to themselves while the experimenter read it out loud. A brief discussion, question and answer period followed. Subjects sat upright in comfortable chairs. Lights were dimmed. Diaphragmatic breathing was demonstrated (Appendix G) and each subject given individual attention as to the proper technique. The experimenter tested the subject's level of relaxation by raising the arm and letting it fall back. Subjects were instructed to silently repeat the word "relax" with each exhalation. Homework was assigned, consisting of practicing the breathing techniques for ten to twenty minutes twice a day, and keeping a daily diary of anxiety-evoking situations and coping or avoidance behavior. Subjects were given copies of their homework contracts and a homework completion chart (see Appendixes J & K).

Session two. This session began with a brief discussion of any problems encountered during home practice. Again, primary emphasis was placed on practicing the diaphragmatic breathing technique and correcting any errors. Subjects practiced slowing down their respiration rate during this session. Subjects were also instructed personally identified anxiety-evoking situation and note disruptions in breathing. This was suggested by Foss (1976) as a way of increasing subjects' awareness of the effect that anxiety-arousing situations have on respiration. The same homework again was assigned.

Session three. This session followed the same format as session two. However, when asked to imagine their feared situation, subjects were asked to try to keep their breathing slow, regular, and deep. As well, subjects practiced diaphragmatic breathing while standing, walking, and jogging in place. This is suggested by Innocenti (1966) as a way of enhancing generalization of diaphragmatic breathing to everyday situations. Discussion and suggestions for continued use of diaphragmatic breathing followed. The same homework was assigned and the second stress-test was individually scheduled.

The control program was also conducted for three, one-hour sessions, and also designated as 'anxiety management training'. Time and attention equivalent to that given to the experimental group were allocated. Subjects received a handout on stress and anxiety exclusive of any specific relaxation instructions (see Appendix H). Subjects were told in essence that "information about anxiety and relaxation will help you to decrease anxiety and increase relaxation". Discussion of anxiety, anxiety-related problems, and non-specific coping strategies took place. Subjects were told that everyone has the capacity to relax but they must let it happen at their own pace. Toward the end of each session subjects were asked to relax as best as they could. Homework consisted of relaxing twice a day on their own, and to keep the anxiety diary described above. Control subjects were also given copies of homework contracts and homework completion charts. The Anxiety Scale (postrelaxation) was administered after the first and last self-relaxation sessions to determine the efficacy of this technique. At the end of the third session subjects were scheduled for the second stress-test.

RESULTS

The research design in this study was a pretest-posttest control group design (Beck, Andrasik, & Arena, 1984), controlling for non-specific variables such as expectancy. Data were analyzed using the Statistical Package for the Social Sciences (Nie, Hull, Jenkins, Steinbrenner, & Brent, 1983), and the Biomedical Data Package (Dixon, W., 1983). Results relevant to the hypotheses are presented first for self-report measures, then for physiological measures. Finally, data collected from the postexperimental questionnaire are presented, providing some sense of how subjects perceived their respective treatments. All analysis of variance tables are shown in Appendix N.

Subject Characteristics

Subjects were first matched according to sex, age, and cigarette consumption, then randomly assigned to either the experimental or control condition. One-way analyses of variance (ANOVAs) performed on age and cigarette consumption confirm that there were no significant differences between groups on these variables. The experimental group consisted of nine subjects (four males, five females), six of whom had received prior treatment for anxiety-related problems. The control group also consisted of nine subjects (three males, six females), five of whom had received prior treatment for anxiety-related problems. Thus, both groups were of similar composition with respect to sex and prior treatment for anxiety problems.

Although subjects were assigned randomly to the two conditions, one-way ANOVAs between groups were computed for all pretest measures to check on their initial comparability. Table 1 presents the data from this analysis. The two groups were comparable on all variables except for the main phobia scale of the Fear Questionnaire, $F(1,16)=6.28$, $p<.05$. This indicated that experimental subjects were more likely to avoid anxiety-evoking situations than were control subjects. Thus, subsequent analysis of this variable includes a covariate adjustment for the difference in initial scores. All self-report measures taken during the pretreatment and posttreatment sessions meet the assumption of homogeneity of variance between groups, as determined by Bartlett-Box F probability values greater than .05.

Credibility of Conditions

Both groups rated their respective programs as highly credible. A preliminary one-way ANOVA computed for Credibility Questionnaire scores revealed no significant difference between groups on the overall score, $F(1,16)=.26$, $p=.62$, (see Table 1). Both groups held equal beliefs in the power and effectiveness of the treatments, $F(1,16)=.59$, $p=.45$ and their expectations for improvement, $F(1,16)=.02$, $p=.88$, and both groups were equally willing to recommend their respective treatments to friends with anxiety

Table 1

Between group comparisons on demographic variables
and initial self-report measures.

MEASURE	EXPERIMENTAL mean	EXPERIMENTAL s.d.	CONTROL mean	CONTROL s.d.	F(1,16)	p
Education (yrs)	13.1	2.8	12.3	2.3	.41	.53
Age	38.7	4.3	34.2	8.3	2.11	.16
Cigarettes/day	19.1	15.1	13.6	19.7	.43	.52
Credibility Questionnaire	25.6	2.9	24.8	3.6	.26	.62
IPAT ASQ	48.1	10.9	55.6	8.6	2.67	.12
Somatic Inventory	64.2	19.2	66.4	24.8	.04	.83
State Anxiety	69.2	7.1	65.1	10.2	.99	.33
Anxiety Scale						
prestress	-2.7	3.1	-.9	2.8	1.68	.21
postrelax.	1.4	3.7	.02	2.5	.90	.36
Fear Questionnaire						
total phobia	47.0	18.3	58.1	21.0	1.43	.25
anx./dep.	21.1	7.8	26.0	7.2	1.92	.18
main phobia	7.0	1.1	5.1	1.9	6.28	.02
global phobia	5.9	2.1	4.7	1.8	1.77	.20

problems, $F(1,16)=.28$, $p=.61$. It is unlikely therefore, that the between-group differences found are due to different levels of expectations.

Self-Report Measures

It was predicted that the experimental condition would be superior to the control condition in decreasing various self-report measures of anxiety. To test this prediction, a 2 (group) x 2 (session) repeated measures ANOVA was computed for each dependent variable. Table 2 presents means and standard deviations for each self-report measure, and the F and p values for the group x session interaction, indicating whether the two treatments differed in impact. In most cases a decrease in score from pretest to posttest indicates a decrease in anxiety as measured by each test. For the Anxiety Scale however, an increase in score indicates a greater degree of relaxation. Analyses revealed significant group x session interaction effects for two variables; the Somatic Inventory, $F(1,16)=8.65$, $p<.05$, and the Anxiety Scale (prestress), $F(1,16)=4.92$, $p<.05$. In partial support of the hypothesis, these findings demonstrate that the experimental condition was more effective than the control condition in; (a) reducing a self-report measure of somatic anxiety, and (b) increasing self-reported relaxation during a stressful situation. Most self-report measures however failed to support the hypothesis of superior effects for the experimental group.

Table 2

Means and standard deviations of self-report variables, and F and p values for the group x session interaction.

VARIABLE	Experimental (n=9) Mean	s.d.	Control (n=9) Mean	s.d.	(G x S) F (1,16)	p
IPAT ASQ						
pre	48.11	10.87	55.67	8.63	2.40	.141
post	43.11	14.08	55.44	10.01		
SOMATIC INVENTORY						
pre	64.22	19.19	66.44	24.87	8.65	.010
post	51.00	25.01	75.00	30.08		
STAI A-STATE						
pre	69.22	7.08	65.11	10.18	1.83	.195
post	47.67	20.15	54.67	10.98		
ANXIETY SCALE (prestress)						
pre	-2.74	3.16	-.91	2.81	4.92	.040
post	-4.48	3.64	2.46	3.37		
ANXIETY SCALE (postrelax)						
pre	1.46	3.75	0.02	2.57	2.48	.135
post	6.79	2.25	3.78	2.98		
FEAR QUESTIONNAIRE						
total phobia						
pre	47.00	18.28	58.11	21.05	.10	.750
post	30.44	11.38	43.56	19.95		
anxiety-depression						
pre	21.11	7.77	26.00	7.19	.02	.890
post	12.00	5.87	17.33	6.18		
*main phobia					F(1,15)	
pre	7.00	1.12	5.11	1.96	1.92	.186
post	4.22	2.49	4.44	1.94		
global phobia						
pre	5.89	2.09	4.67	1.80	2.96	.105
post	3.22	2.22	3.56	1.24		

*includes a covariate adjustment for the significant difference in pretest scores between groups.

Although only two interactions were significant, analyses revealed main effects for the session (pretreatment-posttreatment) factor on the STAI (A-state), the Anxiety Scale (prestress and postrelaxation), and the Fear Questionnaire (total phobia, anxiety-depression, and global phobia scales). These findings suggest that; (a) overall, the treatments were effective in decreasing six of the nine self-report measures, and (b) most of the measures were sensitive to decreases in anxiety level over a short period of time. However, since test reactivity or subjects' expectations could account for these changes between sessions, nothing can be concluded from these results in terms of the relative effectiveness of the two conditions. No analysis was performed on the data obtained from the Anxiety Diaries as compliance was very low for this measure.

Psychophysiological Measures

Two categories of physiological activity were assessed during the laboratory sessions; tonic levels over three periods (baseline, anticipation, and recovery), and specific reactions to the stressor. Tonic levels of activity consisted of respiration rate and depth, heart rate, and frequency of spontaneous skin conductance responses (SCRs). Specific reactions consisted of heart rate accelerations and decelerations, and SCR amplitude and recovery time.

Again, although subjects were randomly assigned to each condition, between group ANOVAs were calculated for initial physiological measures to determine how comparable the groups were at pretreatment. Significant initial differences between groups were found on two physiological measures; thoracic respiration depth, $F(1,16)=6.3$, $p<.05$, and SCR recovery time, $F(1,16)=5.11$, $p<.05$. Subsequent between group analyses for these measures include covariate adjustments for the differences in initial scores.

Analyses were then undertaken to answer two questions, whether: (a) tonic physiological activity increased significantly over baseline levels in anticipation of a loud, piercing noise, and (b) the experimental condition was more effective than the attention control condition in decreasing the various indices of physiological arousal. All physiological measures taken during pretreatment and posttreatment laboratory sessions meet the assumption of homogeneity of variance between groups, as determin[illegible] Bartlett-Box F probability values greater than .05.

Subjective appraisal of the stressor was obtained by a rating from one (not at all) to ten (extremely) of how noxious the subject found it to be. Both groups felt that the noise was moderately distressing as indicated by a mean rating of 7.55 for the experimental group and 7.22 for the control group.

<u>Period Effects</u>. It was expected that anticipation of the stressor (loud tone) would produce increases over baseline levels in tonic physiological activity. Tables 3 to 6 present means and standard deviations of tonic physiological measures across the three periods (baseline, anticipation, and

Table 3

Means and standard deviations of respiration rate for experimental and control conditions across periods and between sessions.

VARIABLE	Experimental (n=9) mean	s.d.	Control (n=9) mean	s.d.
RESPIRATION RATE				
Session 1				
Baseline	22.78	5.80	27.00	4.97
Anticipation	24.00	5.41	26.78	7.03
Recovery	22.22	6.76	28.11	6.58
Session 2				
Baseline	12.56	5.50	23.56	3.21
Anticipation	12.22	5.36	24.00	7.71
Recovery	13.00	6.28	24.89	5.21

Table 4

Means and standard deviations of respiration depth measures for experimental and control conditions across periods and between sessions.

VARIABLE	Experimental (n=9) mean	Experimental (n=9) s.d.	Control (n=9) mean	Control (n=9) s.d.
THORACIC DEPTH				
Session 1				
Baseline	9.67	2.24	6.73	3.75
Anticipation	11.62	3.23	6.69	3.01
Recovery	10.64	2.04	6.67	3.97
Session 2				
Baseline	7.69	3.67	6.35	3.02
Anticipation	8.67	4.33	6.41	2.83
Recovery	8.56	3.93	7.17	3.38
ABDOMINAL DEPTH				
Session 1				
Baseline	3.77	2.50	2.52	2.11
Anticipation	5.21	2.30	2.45	1.89
Recovery	4.45	3.06	2.34	2.00
Session 2				
Baseline	6.09	1.27	2.80	2.17
Anticipation	5.93	1.43	2.63	2.04
Recovery	5.73	1.35	2.81	1.92

Table 5

Means and standard deviations of heart rate measures for experimental and control conditions across periods and between sessions.

VARIABLE	Experimental (n=9) mean	Experimental (n=9) s.d.	Control (n=9) mean	Control (n=9) s.d.
PEAK HEART RATE				
Session 1				
Baseline	83.77	13.60	79.46	13.20
Anticipation	87.52	14.62	80.94	10.85
Recovery	85.41	14.42	80.42	11.71
Session 2				
Baseline	74.16	15.05	73.74	14.05
Anticipation	76.55	14.83	73.09	13.04
Recovery	75.65	15.11	74.46	14.73
LOW HEART RATE				
Session 1				
Baseline	71.05	12.92	69.59	14.51
Anticipation	73.99	13.15	69.17	12.52
Recovery	70.16	12.55	68.06	13.96
Session 2				
Baseline	59.67	15.31	63.94	15.89
Anticipation	61.08	16.43	61.44	14.93
Recovery	61.06	15.70	63.72	15.30

Table 6

Means and standard deviations of the frequency of spontaneous skin conductance responses for experimental and control conditions across periods and between sessions.

Variable	Experimental (n=9)		Control (n=9)	
	mean	s.d.	mean	s.d.
Number of SSCRs				
Session 1				
Baseline	6.67	2.83	6.56	3.54
Anticipation	7.78	3.35	6.33	3.90
Recovery	5.89	2.80	5.[illegible]9	4.04
Session 2				
Baseline	4.11	4.65	2.78	2.44
Anticipation	5.33	3.04	3.67	2.45
Recovery	4.89	3.76	3.00	2.91

recovery). To determine if significant arousal occurred during the anticipation period, a 2 (group) x 2 (session) x 3 (period) repeated measures ANOVA was calculated for each of these measures. Main effects for the period factor were revealed for two measures; thoracic respiration depth, $F(2,32)=3.47$, $p<.05$, and peak heart rate, $F(2,32)=4.55$, $p<.05$. Figures 1 and 2 show changes across periods for thoracic respiration depth and peak heart rate respectively. Although for both measures the experimental group shows a greater increase in arousal during the anticipation period, in neither instance was the period x group interaction significant (see Appendix N). It can be concluded that the effectiveness of the anticipation period in producing arousal was specific to certain measures, with most tonic physiological activity remaining unaffected by the stress manipulation.

Between Group Effects. It was predicted that the experimental condition would show greater decreases than the control condition on measures of physiological activity and reactivity between sessions. To determine whether this was the case, group x session interactions were assessed for each physiological measure (see Appendix N). A significant group x session interaction was found only for respiration rate, $F(1,16)=15.45$, $p<.01$. To confirm that this interaction was indeed a treatment effect, the groups were compared at pretreatment, $F(1,16)=2.40$, $p=.14$, and posttreatment, $F(1,16)=21.22$, $p<.001$. Figure 3 shows mean respiration rates

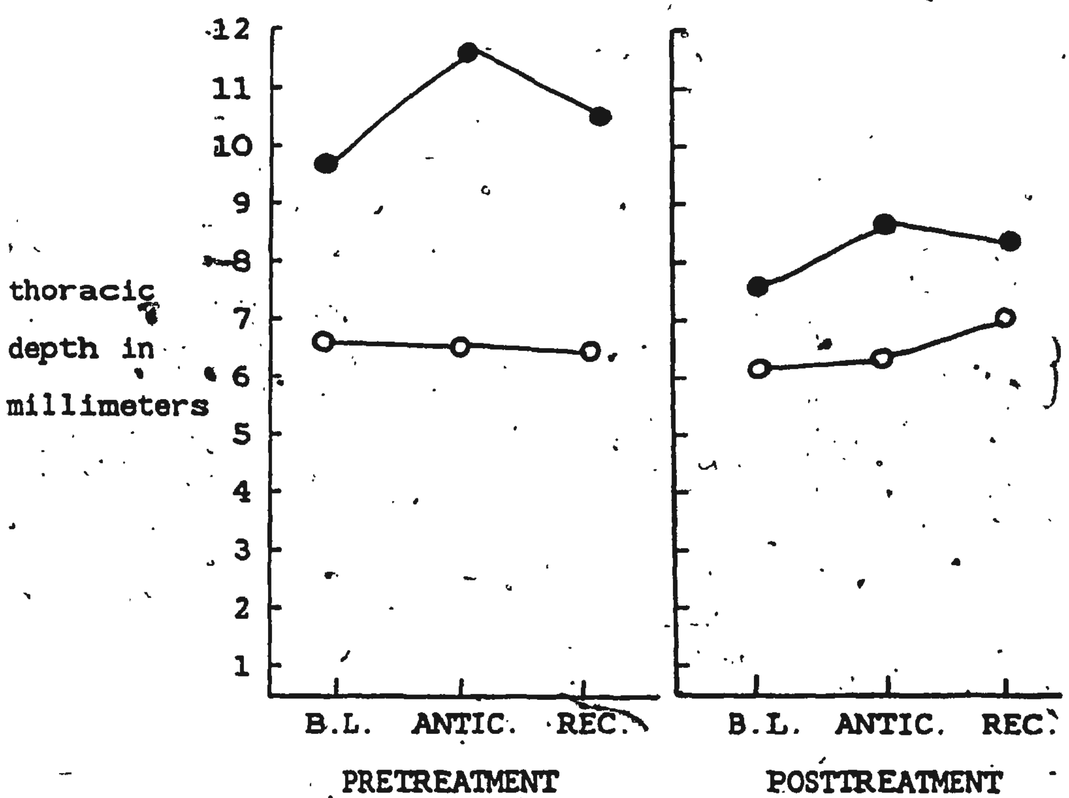

FIGURE 1

Mean thoracic respiration depths for experimental (●——●) and control (o——o) conditions across periods and between sessions.

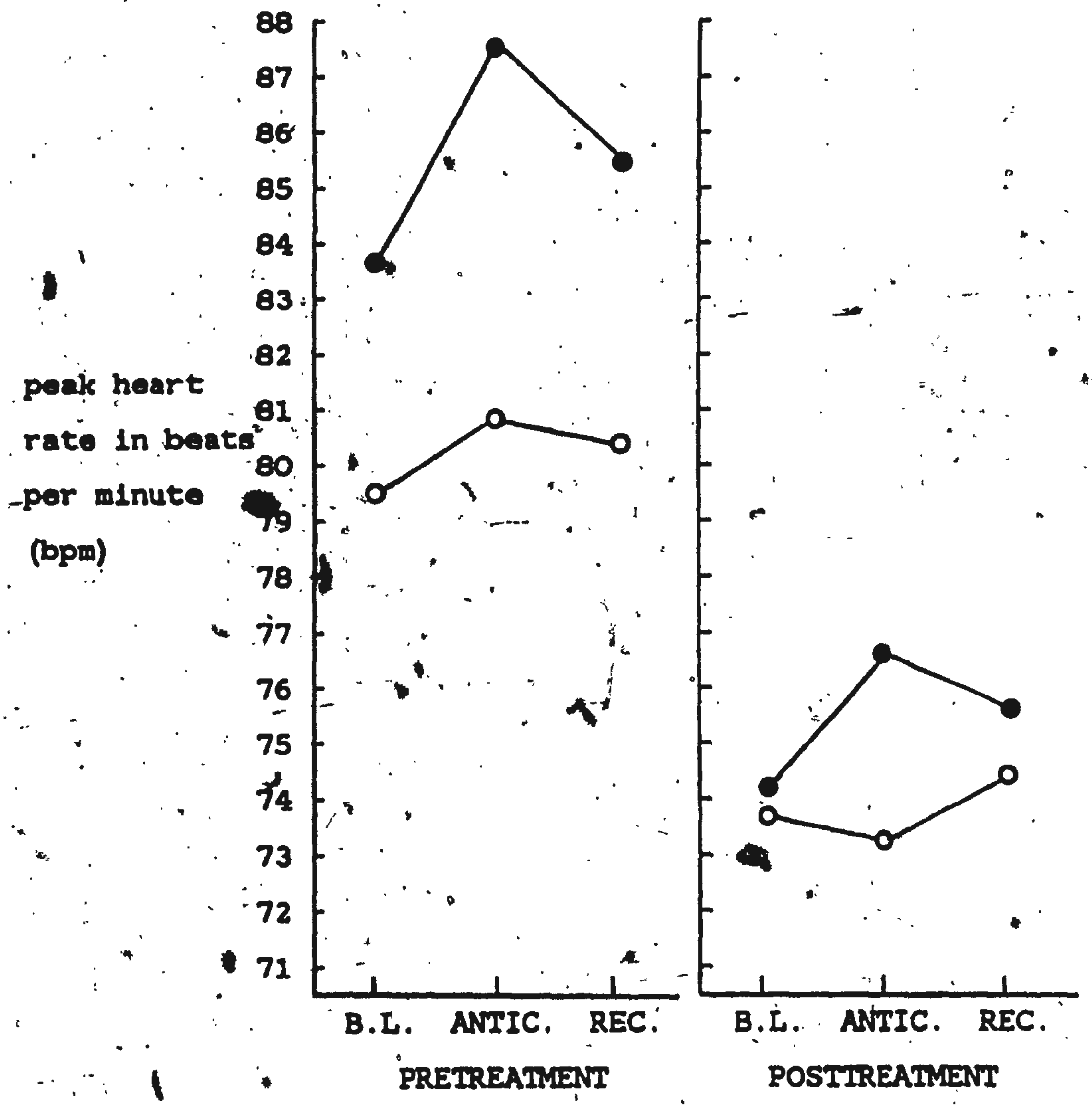

FIGURE 2

Mean peak heart rates for experimental (●——●) and control (o——o) conditions across periods and between sessions.

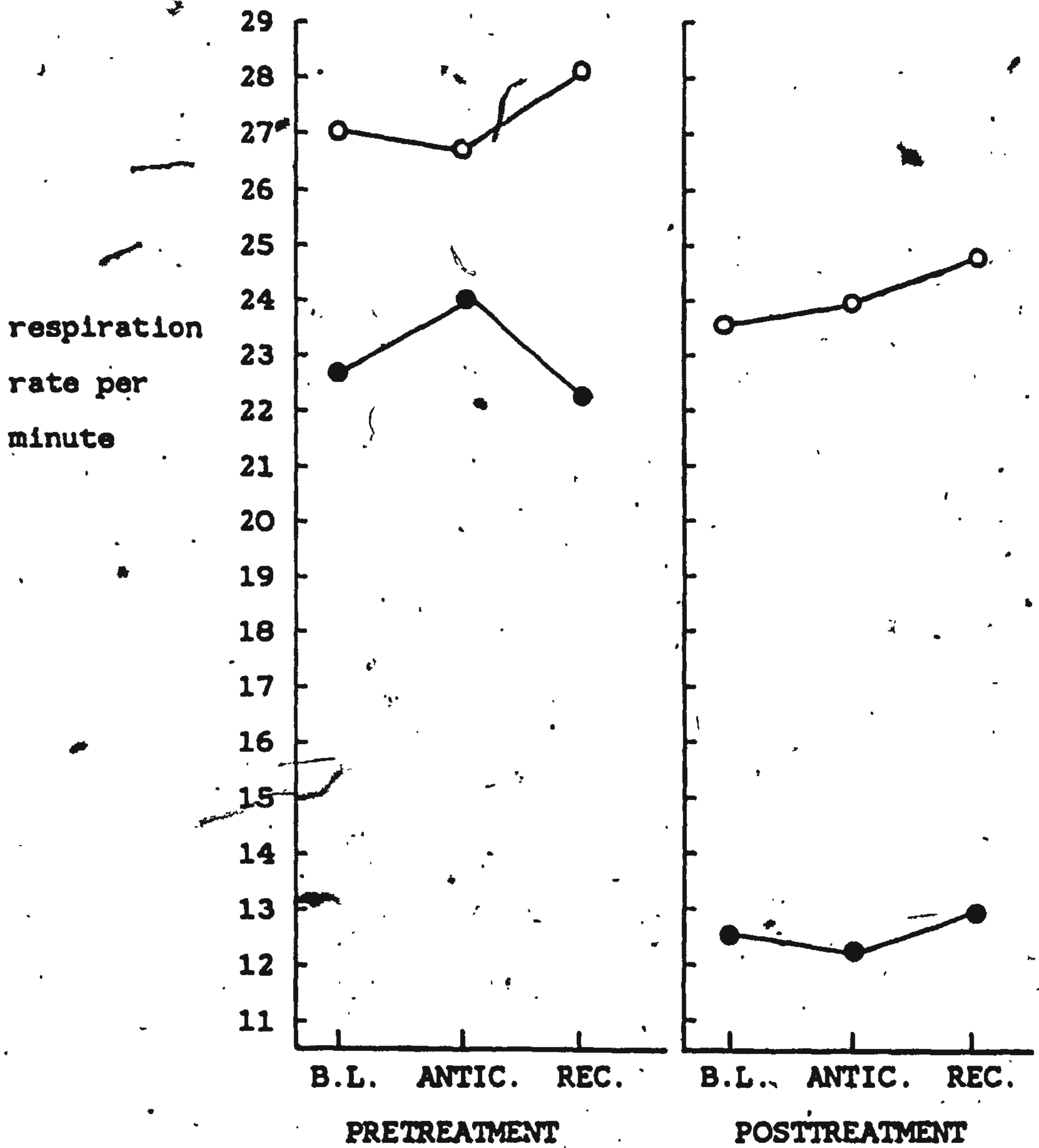

FIGURE 3

Mean respiration rates for experimental (●——●) and control (○——○) conditions across periods and between sessions.

for both groups across periods and between sessions. These results clearly indicate that the superiority of the experimental procedure in decreasing respiration rate. Most measures of tonic physiological activity however failed to support the hypothesis of superior effects for the experimental group.

Table 7 lists means and standard deviations for reactivity measures, and F and p values for group x session interactions. As none of these interactions attained significance, it can be concluded that the experimental condition was not more effective in reducing measures of physiological reactivity to the stressor.

Although only one interaction attained significance, analyses revealed additional main effects for the session (pretreatment-posttreatment) factor on respiration rate, peak heart rate, frequency of SCRs, and heart rate decelerations. Decreases in tonic physiological arousal as measured by respiration rate, peak heart rate, and frequency of SCRs were noted over all subjects. Curiously, although an increase in heart rate deceleration was expected, subjects demonstrated decreases on this measure of physiological reactivity. These findings suggest that for most measures of tonic activity, subjects showed less arousal during the second stress-test. However, neither treatment was effective in reducing specific physiological responses to the stressor itself.

Table 7

Means and standard deviations of physiological reactivity measures and F and p values for the group by session interaction.

VARIABLE	Experimental (n=9) mean	Experimental (n=9) s.d.	Control (n=9) mean	Control (n=9) s.d.	(G x S) F (1,16)	p
HEART RATE RESPONSES						
Accelerations						
Session 1	9.56	9.19	5.33	8.46	1.26	.278
Session 2	3.89	3.65	4.78	6.59		
Decelerations						
Session 1	2.56	4.69	5.33	6.46	0.00	1.00
Session 2	2.33	3.00	.44	5.68		
SKIN CONDUCTANCE RESPONSE						
Amplitude						
Session 1	.76	.43	1.17	.52	.05	.82
Session 2	.59	.38	1.06	.49		
Recovery time						
Session 1	2.62	.66	3.58	1.08	0.00	.97
Session 2	2.53	1.37	3.44	2.80		

Postexperimental Questionnaire

Although data from the postexperimental questionnaire (see Appendix E) were not subject to statistical analysis, group responses to each question are interesting, and are summarized below.

Subjects were asked first what they especially liked about their respective programs. In the breathing therapy group, five subjects stated "the positive effects", two stated "the relaxation periods", one stated "the simplicity of the technique", and one liked the "explanation given for breathing". In the control group, five subjects liked talking about their problems, three stated "the care shown to the clients", and one liked "learning information about anxiety".

When asked what they disliked, seven experimental subjects responded that there was nothing they did not like, one felt that the program was too short, and one did not like the laboratory sessions. Four control subjects responded that there was nothing they did not like, three felt it was too short, one did not like speaking aloud in the group, and one felt there was too much paper work involved.

Six experimental subjects reported that the program met their expectations and three reported that the program exceeded their expectations. Eight control subjects reported that the program met their expectations and one reported that it did not meet her expectations.

The fourth question asked for suggestions to improve the programs. Five experimental subjects made no suggestions for improvement, two felt there should be a followup, one desired more reading material, and one wanted more group discussion. In the control group, five subjects suggested the program be of longer duration, and four made no suggestions for improvement.

Finally, when asked how adequate/professional the therapist was considered to be, subjects on all accounts rated her favorably, from "very adequate" to "excellent".

DISCUSSION

The discussion is presented as follows: discussion of the findings, including alternative explanations of the results; limitations of the study; conclusions; implications for further research; and finally, contributions of this project to treatment of anxiety problems.

Discussion of the Results

The primary hypothesis tested by this research project is that a breathing therapy program is more effective in reducing self-reported anxiety and somatic arousal than an attention control program. To test this hypothesis two equivalent groups were compared on a variety of self-report and psychophysiological measures, before and after three therapy sessions. Differences between groups can confidently be attributed to treatment effects as; (a) both groups held equal expectations regarding the power and effectiveness of their respective programs, and (b) initial values on all variables were controlled by matching, random group assignment, and/or covariate adjustments. Results support the hypothesis that breathing therapy was more effective than the control condition in (a) decreasing respiration rate, (b) reducing symptoms of hyperventilation as measured by the Somatic Inventory, and (c) increasing feelings of relaxation during the laboratory sessions as indicated by the Anxiety Scale.

Essential to the hypothesis is the finding that the breathing therapy program was indeed effective in altering respiration patterns of subjects. Experimental subjects were very successful in slowing their respiration rates during the second stress-test. They also reported greater awareness of their breathing patterns, and increasing comfort with diaphragmatic respiration over the therapy sessions. Although changes in respiration depth measures failed to attain statistical significance, the experimental group did demonstrate an increase in abdominal depth, and a decrease in thoracic depth between sessions whereas control subjects showed virtually no change in depth measures. The experimental manipulation may therefore be assumed to be superior than the control condition in decreasing respiration rate in anxious subjects. It appears clear that this training program waseffective in altering breathing patterns. Since variables such as therapist attention and non-specific relaxation were rigorously controlled, the between group differences in self-report measures discussed below, are most likely attributable to differences in respiration.

Subjects in the experimental condition showed a significant decrease in score on the Somatic Inventory, whereas control subjects showed an increase in score on this measure. According to Lehrer and Woolfolk (1982), a lower score on this inventory indicates a decrease in frequency of symptoms associated with hyperventilation. As most of the items on this inventory are not obviously associated with respiration, it is unlikely that subjects' expectations regarding breathing therapy influenced their scores. It is more probable that changes in subjects' respiration patterns were instrumental in decreasing these symptoms.

According to scores on the Anxiety Scale, experimental subjects also showed a superior ability to relax during a moderately stressful situation (stress-test) using their breathing techniques. It may be that slower, deeper respiration enhances relaxation by decreasing physiological cues associated with anxiety. Also, by consciously focusing on the breathing process, one may be less likely to attend to anxiety-arousing cognitions which often occur in stressful situations.

It was also hypothesized that anticipation of a stressor (loud tone) would produce arousal over baseline levels on measures of tonic physiological activity. The expectation was that physiological effects of breathing therapy would be revealed more clearly during a stressful situation (Lehrer, 1978). Unfortunately however, no firm conclusions can be drawn from the data regarding the efficacy of the anticipation period in producing significant physiological arousal. Results were variable, with most measures showing very little change over the three periods. Period effects were shown only for thoracic respiration depth and heart rate measures. However, these changes were not consistent between groups, and sometimes reflected a decrease in arousal instead of the expected increase. There are several explanations for the general lack of significant arousal over periods. Possibly, as shown by other investigators (Harris et al., 1976; Holmes et al., 1978), the threat of electric shock is more effective in inducing physiological arousal. Although subjects indicated that they found the loud tone to be moderately noxious, they may have reacted more to anticipation of an electric shock. A more likely explanation is that subjects were anxious to begin with. It is probable that being wired up to polygraph equipment in a closed, unfamiliar laboratory was a sufficiently stressful situation in itself, possibly causing higher baseline levels of physiological activity. According to Wilder (1957) the higher the initial level of physiological activity, the smaller the arousal in response to stress. As subjects' baseline levels of tonic activity did appear to be fairly high, perhaps a longer acclimation period would have lowered these levels, permitting greater arousal during anticipation. It remains uncertain however whether greater arousal during the anticipation period would have increased differentiation of treatment effects.

Main effects for the session (pretreatment-posttreatment) factor were found for several self-report and physiological variables. For all of these measures except one (heart rate deceleration), the changes between sessions reflect a decrease in anxiety/arousal for subjects in both groups.

According to the Anxiety Scale, subjects in both conditions were able to relax more before the second stress-test, compared to the initial one, and during the last therapy session, compared to the first. Thus, although the breathing technique produced a greater degree of relaxation during a stressful situation (stress-test), it was not more effective in promoting relaxation in a non-stressful environment (relaxation sessions). Although the increase in self-reported relaxation could be due to familiarity with each situation, or to subjects' expectations of improvement, it is also probable that both groups were able to relax using their respective techniques. Control subjects, although not given detailed instructions, were likely able to relax by just sitting quietly, especially since they had practiced doing this twice daily for two weeks. Findings by Pollak and Zeiner (1979) support the contention that just sitting quietly can promote relaxation. It may also be that control subjects devised their own effective relaxation techniques although information about this was not elicited.

Significant decreases in scores were also found over all subjects on the State Anxiety Scale, and the total phobia, anxiety-depression, and global phobia scales of the Fear Questionnaire. For the most part, these changes reflect an increase in ability to cope with anxiety-evoking situations. As very few subjects filled out the Anxiety Diaries however, it was not possible to compare these self-reports with actual coping behaviors.

Physiological measures showing significant between session reductions consisted of respiration rate, peak heart rate, and frequency of SCRs. These decreases probably reflect less anxiety regarding the nature of the stress-test, and correspond to self-reports of greater relaxation during the second laboratory session.

Many self-report and physiological measures failed to differentiate the groups, or to show expected changes between sessions. It may be informative to examine why hypothesized results did not occur.

Both abdominal and thoracic respiration depth measures failed to differentiate the groups at posttreatment. Due to the overlearned nature of respiration patterns, perhaps it is unreasonable to expect the magnitude of depth change that would yield statistical significance in just three therapy sessions. Lum (1976) has reported that it may take up to a year for effective alteration of thoracic breathing. Perhaps then, greater depth changes would have been attained with a longer program. It may also be that the decrease shown in respiration rate among experimental subjects was insufficient to effect a concomitant change in depth. Perhaps a slower respiration rate (7 or 8 cycles per minute versus 12) would have produced a compensating increase in abdominal respiration depth. It is also possible that, had the change in respiration depth been greater for the experimental group, a stronger heat rate effect would have occurred. Sroufe (1971) and Wood and Obrist (1964) have demonstrated that it is respiration depth, not rate, which exerts a primary influence on heart rate.

Cardiac accelerations to stimuli are typically interpreted as components of a defensive reflex, whereas cardiac decelerations are interpreted as components of an orienting reflex to stimuli (Graham and Clifton, 1966). The results for cardiac decelerations however, were unexpected.

As subjects were subjectively less anxious and showed lower levels of physiological arousal during the second stress-test, it was expected that they would show a greater decelerative response to the stimulus. Lehrer et al. (1980) state that the emission of an orienting reflex in response to a stressful stimulus may reflect the fact that the person has a strategy for coping with the stress. This apparently was not the case since overall, a significant decrease in heart rate deceleration occurred between sessions. Perhaps as Lehrer (1978) suggests, chronic anxiety inhibits the person from emitting orienting reflexes to stressful situations. Indeed for both groups the predominant cardiac response for each session was acceleration. Another possible explanation is that the lowest poststimulus heart rate used in the calculation of deceleration was taken within ten seconds after stimulus onset. Possibly a greater decelerative response would have occurred if this ten second period had been extended. As Lang and Hnatiow (1962) point out, an accelerative response occurs almost immediately after stimulus onset whereas a decelerative response continues for as far as twenty pulses after stimulus onset.

Finally, the lack of effect of respiratory modification on the SCR is surprising given Harris et al.'s (1976) demonstration of reduced SCR amplitude to stress for slow breathing subjects. However again it must be noted that Harris et al. (1976) used nonanxious college students in contrast to the anxious subjects in this study. It seems probable that habituation to a stimulus is slowed or prevented by high levels of anxiety, (Lader and Wing, 1966).

It is difficult to draw definitive conclusions from the results of the postexperimental questionnaire. Overall, it appears that experimental subjects rated their program slightly higher than control subjects rated theirs. However, in all but one case, both programs met or exceeded subjects' expectations. Control subjects generally felt that their program should be of longer duration, whereas most of the experimental subjects reported benefits after three sessions. This may be [illegible] the different nature of each program; one being hi[illegible] structured, the other (control) less so. Both groups rated the therapist very favorably. This decreases the liklihood that therapist expectations influenced the outcomes of this study.

Limitations of the Study

Several limitations of this study are apparent, decreasing the strength of results and of conclusions which may be drawn. First, it may be that the program was not long enough or intensive enough, to produce significant treatment effects on many variables. Given the anxious nature of the subjects, it is probable that more practice of the breathing techniques would have enhanced treatment effects.

Followup assessment of subjects who took part in this study would have provided some useful information. A followup could have determined how likely subjects were to continue to use their respective techniques. It would have shown if diaphragmatic breathing was a useful coping technique in the long term, and if it became the habitual mode of respiration.

Finally it may be that experimenter bias was not completely controlled as both groups were conducted by one person who was not blind to the nature of the study. It is possible that the researcher induced greater expectations for improvement in the experimental group than in the control group. However, this seems unlikely given the equal credibility ratings of the conditions and the equal therapist ratings on the postexperimental questionnaire.

Conclusions

It may be concluded that the breathing therapy program was more effective than self-relaxation in decreasing respiration rate and certain self-reported indices of anxiety. Although most of the measures used in this study did not demonstrate the superiority of the experimental condition, in no instance was the attention control condition superior. It does appear however that non-specific elements of therapy have a beneficial effect in reducing anxiety as well. As none of the physiological measures, other than respiration rate, differentiated between the two treatments, it may be that certain self-report measures are more sensitive to changes in anxiety levels over brief periods of time. This is consistent with findings of other investigators (Clark, 1978; Mathews, 1971) that there is a considerable degree of dissociation between physiological, behavioral, and subjective changes during treatment. Mathews (1971) postulates that separate components of anxiety tend to change in differing amounts at different times. It is probable that, had this research program been more intensive, and/or of longer duration, greater changes in respiration rate and depth would have occurred, and the effectiveness of breathing therapy been more evident.

Implications for Research

Several opportunities for further research are suggested by this study. Most importantly, research needs to be done to test the continued effectiveness of breathing therapy programs over time. This could be accomplished by an extended followup of subjects or clients receiving diaphragmatic breathing instructions. It is felt that, since the techniques are easy and reportedly enjoyable to implement, people will persist in using them. However, this must be established empirically.

More extensive breathing therapy programs may be necessary to institute long-standing changes in respiration rate and depth. Studies of visual or auditory feedback of respiration patterns to improve training would be a useful addition to the literature in this area. Holmes et al. (1978) showed that normal subjects were able to replicate relaxed breathing patterns (rate and depth) shown to them on a video monitor. A similar technique may be an effective adjunct to breathing therapy programs for anxious clients.

It would be useful to compare breathing therapy to other types of anxiety management techniques such as medication and progressive muscle relaxation. There is no apparent risk of harmful side effects with the use of breathing therapy as there is with medication, and clients may find it easier to learn and implement than progressive muscle relaxation. Breathing therapy programs could also be conducted with various populations to determine differential effects of this intervention. For example, chronic hyperventilators may benefit more from this technique than would those with specific phobias. Another potentially beneficial application of diaphragmatic respiration is as an alternative to maladaptive, anxiety-induced behaviors such as smoking, bruxism, and nail biting.

Finally, Grossman (1983) cites evidence relating slow, diaphragmatic respiration to cardiovascular health. Therefore, breathing therapy could have important implications in preventative medicine. Longitudinal research may reveal that teaching at-risk populations the principles of proper breathing decreases chances of cardiovascular disease (cf Grossman, 1983).

Contributions to Behavior Therapy

This study also has some implications for the practice of behavior therapy. By paying attention to an involuntary behavior such as breathing, a therapist may be able to acquire some useful information about his/her client. If a client is extremely anxious, the therapist can quickly teach him/her to alter his/her respiration, bringing anxiety under control. This may help establish rapport and make a client more amenable to further therapy. Thus diaphragmatic breathing may be used alone or in conjunction with other behavioral techniques. Breathing therapy helps individuals attend to the internal, physical cues of anxiety, especially breathing, rather than a specific anxiety-arousing stimulus. Therefore it lends itself well to group treatment of individuals with diverse anxiety-related problems. Finally, as it is a simple technique to learn and teach, breathing therapy can be efficiently taught by trained paraprofessionals.

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Appendix A

THE ANXIETY SCALE

Check the phrase which best describes how you have been feeling during the past few minutes. Check only one.

GREAT

SURE

CALM

OKAY

INDIFFERENT

UNSURE

SHAKY

FRIGHTENED

SCARED TO DEATH

Check the phrase which best describes how you have been feeling for the last few minutes. Check only one.

REALLY GREAT

CONFIDENT

CONTENT

COOL

NOT TOO BAD

DOUBTFUL

UPSET

FEARFUL

TERRIFIED

Check the phrase which best describes how you have been feeling during the last few minutes. Check only one.

AT EASE

COLLECTED

SO SO

CONCERNED

UNEASY

TENSE

SCARED

PANICKY

Appendix B

SOMATIC INVENTORY

Score each of the following symptoms on a scale from 1 to 9 indicating its frequency of occurrence; 1 indicates that it never happens, 9 indicates that the symptom is almost always present.

1. I can't catch my breath.

   1 2 3 4 5 6 7 8 9

2. I breathe rapidly.

   1 2 3 4 5 6 7 8 9

3. I feel dizzy.

   1 2 3 4 5 6 7 8 9

4. I experience chest pains.

   1 2 3 4 5 6 7 8 9

5. My arms or legs feel weak.

   1 2 3 4 5 6 7 8 9

6. My stomach hurts.

   1 2 3 4 5 6 7 8 9

7. I experience muscular aches and pains.

   1 2 3 4 5 6 7 8 9

8. My muscles twitch or jump.

1 2 3 4 5 6 7 8 9

9. My arms or legs feel stiff.

1 2 3 4 5 6 7 8 9

10. I have difficulty swallowing.

1 2 3 4 5 6 7 8 9

11. My limbs tremble.

1 2 3 4 5 6 7 8 9

12. I feel numbness in my face, limbs or tongue.

1 2 3 4 5 6 7 8 9

13. My neck feels tight.

1 2 3 4 5 6 7 8 9

14. My heart pounds.

1 2 3 4 5 6 7 8 9

15. I experience a tingling sensation somewhere in my body.

1 2 3 4 5 6 7 8 9

16. My throat gets dry.

1 2 3 4 5 6 7 8 9

Appendix C

ANXIETY DIARY

INSTRUCTIONS: Each day, please indicate at least one situation in which you feel anxious, uptight or stressed. Indicate also how you felt and how you coped with the situation. Finally, rate yourself on a scale of one to ten how well you feel you dealt with the situation. One indicates that you dealt poorly with the situation, ten indicates that you dealt very well with it.

EXAMPLE:

Situation: Standing in line at the bank.

Reaction: Close to panic.

Way of coping: I left.

Scale: (1) 2 3 4 5 6 7 8 9 10

Day 1

Scale: 1 2 3 4 5 6 7 8 9 10

Appendix D

CREDIBILITY QUESTIONNAIRE

from Borkovec & Nau (1972)

1. How powerful and effective do you believe this treatment to be?

   1 2 3 4 5 6 7 8 9 10

2. How much improvement do you expect in your ability to cope with stress?

   1 2 3 4 5 6 7 8 9 10

3. How willing would you be to recommend this treatment to a friend with stress problems?

   1 2 3 4 5 6 7 8 9 10

Appendix E

POSTEXPERIMENTAL QUESTIONNAIRE

1. If anything, what did you especially like about the program, from initial interview to stress-tests, to therapy sessions?

2. If anything, what did you dislike about the entire program?

3. Did the therapy sessions meet/exceed/not meet your expectations? How?

4. Do you have any suggestions for improvement of any aspect of this program?

5. How adequate/professional did you consider the therapist to be?

6. Would you like to be referred for additional therapy programs? If so, specify the area:

   a. assertion training
   b. social skills training
   c. agoraphobia
   d. further relaxation training

Appendix F

ANXIETY MANAGEMENT TRAINING MANUAL

> "A harmonious mind matches a rather slow and regular respiration; a troubled psyche speeds up the heart and breathing rhythms. All of us have experienced occurrence whenever we pant or are short of breath because of some sudden emotion. The statement is reversible: a slow, harmonious conscious breathing greatly promotes the mental faculties and the relaxtion of the mind."
>
> \- S. Brena (1972)

Anxiety and tension are an inherent part of living in today's stressful world. Although it is neither possible nor desirable to totally get rid of these feelings, it is possible and desirable to decrease their frequency and intensity. This is especially true for people who respond to many situations with a great deal of anxiety. Anxiety reactions are also known as the "fight or flight" response. That is, certain bodily changes occur which prepare us to deal with "dangerous" or stressful situations by either fighting or running away as fast as we can. As most of us have experienced, during this response, our hearts pound, respiration increases and we may get a feeling of breathlessness, our palms sweat and in general, we feel a sense of very high arousal or panic. This is an "adaptive" response as it enabled our cavemen ancestors to effectively deal with life-threatening situations. However, in our everyday lives we rarely have the opportunity to fight back in or run away from stressful situations. Rather we often must pretend to remain calm, cool and collected while inside we are churning with emotion although unable to act to expel this pent up energy. This is not only a very uncomfortable state to be in, it may be dangerous as well as it causes alot of wear and tear on the body especially if it happens frequently. This stress response may cause a wide variety of disorders including ulcers, high blood pressure and cardiac trouble. As a matter of fact, it is reported that two-thirds of the office visits to family doctors are prompted by stress-related symptoms.

Since many of the reactions involved in anxiety are physical, it makes sense that if we could control these responses, we could control our anxiety. Unfortunately it is practically impossible to "will" our hearts to slow down, or to stop a blush. However, it is very easy to bring our respiration under voluntary control. Although breathing is usually automatic (we don't need to think to take a breath!), we can change it just by giving ourselves a command....try it....take a deep breath, sniff, sigh.

Voluntary control of respiration patterns (breath control) is perhaps the oldest stress-reduction technique known. It has been used for thousands of years to reduce anxiety and to promote a generalized state of relaxation. Voluntary breath control is an important part of Hatha Yoga, Zen Meditation, Kung-fu and more recently progressive relaxation techniques and natural childbirth. Important advantages of using this natural method of stress control are ease of learning, freedom from potential side-effects often caused by medication, and its availability. Voluntary breath control can be used anytime, anywhere.

There are three types of breathing patterns which are called clavicular, thoracic and diaphragmatic. Clavicular and thoracic breathing are shallow types of respiration allowing air into only the upper parts of the lungs. This is not very efficient and results in more rapid respiration to obtain sufficient oxygen. However, it has been noted that thoracic respiration is probably the most common type. It also has been shown that thoracic, or shallow, rapid breathing is related to feelings of anxiety and it may even bring about the fight or flight response.

We shall be concerned with learning diaphragmatic breathing. This is the deepest and most efficient type of respiration. It allows oxygen to get to the lowest parts of the lungs where it is efficiently distributed throughout the body via the bloodstream. During the diaphragmatic breath, the diaphragm (a thin, muscle-like structure which separates the thoracic and abdominal cavities) flattens downward on inhalation. This forces air to descend into the lungs and at the same time it forces the organs in the abdomen to be pushed down and forward. Research evidence shows that this type of respiration is associated with a relaxed state of mind and may also be capable of inhibiting the fight or flight response. Although infants and small children primarily use their diaphragms in breathing, many adults have gotten out of the habit, perhaps due to reluctance in letting the abdomen protrude. Tight pants, skirts and girdles also inhibit the ability to breathe deeply and smoothly. Some therapists feel that restricted diaphragmatic movement is due to bottling up emotions inside. Indeed, expressing emotions is associated with deep breathing and diaphragmatic movement as reflected by statements such as "sigh of relief", "belly laugh" and "heaving with grief".

A major part of this program consists of practicing the breathing techniques which you will learn. It must be kept in mind that the way in which you breathe is a very deeply ingrained habit. You breathe approximately twenty-two thousand times every day! However, with conscious effort, daily practice and group support you will be able to learn a more adaptive, relaxed manner of respiration which will help you reduce general anxiety and decrease the severity of your responses to stressful situations. Look at your daily relaxation breaks as gifts to yourself, your health and well-being.

Appendix G

DIAPHRAGMATIC BREATHING INSTRUCTIONS

There are several things to keep in mind when practicing your breathing exercises. Don't worry if the techniques seem awkward at first, with practice they will become automatic. Below are listed some helpful hints which you will find useful and which should be incorporated into your daily practice.

1. The place in which you practice your relaxation exercises is important. It should be well ventilated and free from all distractions. If necessary put a sign on the door and unplug the phone. You should try to practice in the same place and at the same times each day.

2. You should not practice soon after a meal, a cigarette or coffee/tea/cola.

3. Practice ten to twenty minutes twice a day.

4. Don't wear restricting garments.

5. The main object is to transfer most of your breathing to the abdomen, so that on quiet respiration most of the movement takes place in the abdomen and very little movement of the thorax (chest) occurs. Place one hand on the abdomen, the other on the chest. When you inhale, the abdomen should swell into your hand whereas the hand on the chest should remain still.

6. The rhythm is important. Inhalation should flow smoothly into a relaxed exhalation. Exhalation should last slightly longer than inhalation. There should be a brief pause after exhalation.

7. Exhalation should never be forced. Say "relax" to yourself as you exhale.

8. Work on technique first. When you have this down pat, try to decrease your rate of breathing. Ideal rate is different for everyone but should be around eight breaths per minute.

9. Check your breathing at all possible opportunities throughout the day. Check (or have someone check for you) for unconscious mannerisms such as sniffing, sighing and gasping.

10. Use this technique as a coping strategy whenever you begin to feel anxious, or during a stressful situation.

11. As you practice your exercises you may find that you are either carried away by your thoughts or you are making a deliberate attempt not to think. It is best to observe your thoughts passively and then let them float on by and gently redirect your awareness to the breathing process.

Appendix H

ANXIETY MANAGEMENT TRAINING MANUAL (C)

Anxiety and tension are an inherent part of living in today's world. Although it is neither possible nor desirable to totally get rid of these feelings, it is possible and desirable to decrease their frequency and intensity. This is especially true for people who respond to many situations with a great deal of anxiety. Anxiety reactions are also known as the "fight or flight" response. That is, certain bodily changes occur which prepare us to deal with "dangerous" or stressful situations by either fighting or running away as fast as we can. As most of us have experienced, during this response our hearts pound, our palms sweat and in general, we feel a sense of very high arousal or panic. This is an "adaptive" response as it enabled our cavemen ancestors to effectively deal with life-threatening situations. However, in our everyday lives we rarely have the opportunity to fight back in or run away from stressful situations. Rather we often must pretend to remain calm, cool and collected while inside we are churning with emotion although unable to act to expel this pent-up energy. This is not only a very uncomfortable state to be in, it may be dangerous as well as it causes alot of wear and tear on the body, especially if it happens frequently. This stress response may lead to a wide variety of disorders including ulcers, high blood pressure and cardiac trouble. As a matter of fact, it is reported that two-thirds of the office visits to family doctors are prompted by stress-related symptoms.

Possibly one of the easiest and most effective techniques of stress management is to simply identify and keep track of stress-promoting situations and note how we react to them. Becoming aware of these situations is the first step in overcoming their impact. When you know which situations are stressful, you increase your motivation to do something about how you react to them. How we deal with these situations then depends both on the situation itself and our own coping styles. These strategies may be simple or complex, but there are virtually unlimited strategies available to the imaginative mind.

People with an anxious personality suffer from anxiety to a far greater extent than other people because their reaction to stress results in a form of anxiety which seems to perpetuate itself. In so-called normal individuals, the fight or flight response subsides soon after the stressor is removed. However, anxious persons tend to have a more severe form of anxiety reaction which may pose a greater threat to their health and well-being. The anxious individual suffers from an anxiety "feedback loop" that perpetuates the anxiety reaction. Any arousal response to some stressor can eventually become a stressor itself and in turn cause further arousal. Basically this feedback is cognitive, in the form of thoughts concerning the nature of the stressor and possible outcomes. These fear laden thoughts themselves may bring on the fight or flight response. Two examples of this type of thinking are "catastrophizing", where one thinks of the worst possible outcome of a situation, and "reliving", where one thinks about a situation over and over again, each time experiencing an anxiety reaction.

As we can see then, our personality, the way we perceive and evaluate information has alot to do with how we react to situations. Few events are innately stressful, but we make them stressful by the way in which we perceive them. A person may alter these stress-causing attitudes by first becoming aware of them and then working to change them. This is known as personality engineering.

Finally, learning to relax is an important component of anxiety management. Relaxation has been shown to produce a lasting reduction in stress-related symptoms if practiced for ten to twenty minutes twice a day. Relaxation decreases physical arousal to stressors. There are many ways to relax but each method has in common removing yourself from all sources of disturbance and just sitting quietly. No specific technique has been proven to be superior. Many people are uncomfortable with quietness, having been bombarded with stimulation every waking minute of their lives. They have not learned how to just sit back and relax. The best way to relax is just to let it happen at its own pace, maintaining a passive attitude. Everyone is capable of this skill.

In summary then, this program is aimed at helping you deal with tension and anxiety by using three effective techniques; keeping track of anxiety-arousing situations, group discussion and learning to relax. Each technique will require some effort on your part, but look at it as a gift to yourself, your health and well-being.

Appendix I

ANXIETY MANAGEMENT TRAINING CONTRACT

As a requirement for admission to the Anxiety Management Program I, ______________ hereby agree to the following terms:

1. I will attend all three therapy sessions.
2. I will do the homework assignments to the best of my ability.
3. I will complete any questionnaire given to measure my progress.
4. I will undergo two "stress-tests".
5. I will deposit $10.00, refundable upon completion of the program.

______________________ signature ______________________

date

Appendix J

HOMEWORK CONTRACT

I, ______________ agree to practice relaxing for 10 to 20 minutes two times per day. I will practice: ______________ (place) at ________ and ________ (times) each day.

In addition, I will keep a diary when and where I feel anxious and how I deal with the situation.

signature

HOMEWORK CONTRACT

I, ______________ agree to practice my breathing techniques for 10 to 20 minutes two times per day. I will practice ______________ (place) at _______ and _______ (times) each day.

In addition, I will keep a diary of when and where I am anxious and how I deal with the situation.

signature

APPENDIX K

Relaxation Exercise

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8

Day 9	Day 10	Day 11	Day 12	Day 13	Day 14	Day 15	Day 16

Anxiety Diary

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8

Day 9	Day 10	Day 11	Day 12	Day 13	Day 14	Day 15	Day 16

ASSIGNMENT COMPLETION CHART

Place a star (*) in the appropriate box each time you practice your relaxation exercise (twice a day) and each time you fill out your Anxiety Diary (at least once a day).

Appendix L

INDIVIDUAL DATA SHEET

Name: ____

Age: ________ Phone number: ________

Male: ________ Female: ________

Please list any medication and dosage you are currently taking. Type: ________________ Dose: ____________

________________ ____________

________________ ____________

Do you smoke cigarettes? Yes: ______ No: ______ If so, how many do you smoke per day? ____________

Have you ever had any experience with formal relaxation training, yoga, meditation or natural childbirth? If so, describe: ______________________________

____

Do you have any heart or respiratory disorders? _____

____

Have you taken any major tranquilizers in the past six months? ______________________

Have you ever been treated for anxiety problems? ______

____

____

Appendix M

Instructions for stress-tests

Pretreatment and posttreatment control group instructions. "Soon you will hear a soft tone from the speakers placed behind your head. This is a warning signal. Sometime after this tone you will hear a very loud, piercing burst of noise. Please try to remain as calm and relaxed as possible."

Posttreatment experimental group instructions. "Soon you will hear a soft tone from the speakers placed behind your head. This is a warning signal. Sometime after this tone you will hear a very loud, piercing burst of noise. Please try to remain as calm and relaxed as possible by regulating your breathing the way you have been taught. Remember, keep it even, deep and slow."

Appendix N

Analysis of Variance Tables

1. Oneway analysis of variance between groups on initial self-report and demographic variables.

2. Two (group) by two (session) repeated measures analysis of variance on self-report variables using sequential sum of squares.

3. Two (group) by three (period) repeated measures analysis of variance on session one measures of tonic physiological activity.

4. Oneway analysis of variance between groups on initial measures of physiological reactivity.

5. Two (group) by two (session) by three (period) repeated measures analysis of variance on measures of tonic physiological activity.

6. Two (group) by two (session) repeated measures analysis of variance on measures of physiological reactivity using sequential sum of squares.

ONEWAY ANALYSIS OF VARIANCE BETWEEN GROUPS ON INITIAL SELF-REPORT AND DEMOGRAPHIC MEASURES.

EDUCATION (YEARS)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	2.72	1	2.72	.41	.532
WITHIN GROUPS	106.89	16	6.68		
TOTAL	109.61	17			

AGE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	93.39	1	93.39	2.11	.165
WITHIN GROUPS	707.11	16	44.19		
TOTAL	800.50	17			

NUMBER OF CIGARETTES PER DAY

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	133.39	1	133.39	.43	.520
WITHIN GROUPS	4944.89	16	309.06		
TOTAL	5078.28	17			

IPAT

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	256.89	1	256.89	2.67	.122
WITHIN GROUPS	1540.89	16	96.31		
TOTAL	1797.78	17			

SOMATIC INVENTORY

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	22.22	1	22.22	.04	.835
WITHIN GROUPS	7897.78	16	493.61		
TOTAL	7920.00	17			

STATE-TRAIT ANXIETY INVENTORY (A-STATE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	76.06	1	76.06	.99	.335
WITHIN GROUPS	1230.44	16	76.90		
TOTAL	1306.50	17			

ANXIETY SCALE (PRESTRESS)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	14.94	1	14.94	1.68	.214
WITHIN GROUPS	142.62	16	8.91		
TOTAL	157.56	17			

ANXIETY SCALE (POSTRELAXATION)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	9.24	1	9.24	.89	.358
WITHIN GROUPS	165.14	16	10.32		
TOTAL	174.39	17			

FEAR QUESTIONNAIRE (TOTAL PHOBIA)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	555.56	1	555.56	1.43	.249
WITHIN GROUPS	6218.89	16	388.68		
TOTAL	6774.44	17			

FEAR QUESTIONNAIRE (ANXIETY-DEPRESSION)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	107.56	1	107.56	1.92	.185
WITHIN GROUPS	896.89	16	56.06		
TOTAL	1004.44	17			

FEAR QUESTIONNAIRE (MAIN PHOBIA)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	16.06	1	16.06	6.28	.023
WITHIN GROUPS	40.89	16	2.56		
TOTAL	56.94	17			

FEAR QUESTIONNAIRE (GLOBAL PHOBIA)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	6.72	1	6.72	1.77	.202
GROUPS	60.89	16	3.81		
TOTAL	67.61	17			

TWO (GROUP) BY TWO (SESSION) REPEATED MEASURES ANALYSIS OF VARIANCE ON SELF-REPORT VARIABLES USING SEQUENTIAL SUM OF SQUARES.

IPAT

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	342.78	16	21.42		
SESSION	61.36	1	61.36	2.86	.110
GROUP BY SESSION	51.36	1	51.36	2.39	.141

SOMATIC INVENTORY

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	1972.89	16	123.31		
SESSION	49.00	1	49.00	.39	.537
GROUP BY SESSION	1067.11	1	1067.11	8.65	.010

STATE ANXIETY SCALE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	2424.22	16	151.51		
SESSION	2304.00	1	2304.00	15.21	.001
GROUP BY SESSION	277.78	1	277.78	1.83	.195

ANXIETY SCALE (PRESTRESS)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	108.77	16	6.80		
SESSION	252.28	1	252.28	37.11	.000
GROUP BY SESSION	33.45	1	33.45	4.92	.041

ANXIETY SCALE (POSTRELAXATION)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	36.11	16	2.26		
SESSION	185.87	1	185.87	82.35	0.000
GROUP BY SESSION	5.60	1	5.60	2.48	.135

FEAR QUESTIONNAIRE (TOTAL PHOBIA SCALE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	1371.22	16	85.70		

SESSION	2177.78	1	2177.78	25.41	.000
GROUP BY SESSION	9.00	1	9.00	.10	.750

FEAR QUESTIONNAIRE (ANXIETY-DEPRESSION SCALE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	402.44	16	25.15		
SESSION	711.11	1	711.11	28.27	.000
GROUP BY SESSION	.44	1	.44	.02	.896

FEAR QUESTIONNAIRE (MAIN PHOBIA SCALE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
COVARIATES					
FQMP1	12.49	1	12.49	3.13	.097
MAIN EFFECTS					
GROUP	7.68	1	7.68	1.92	.186
EXPLAINED	20.16	2	10.08	2.53	.113
RESIDUAL	59.83	15	3.99		
TOTAL	80.00	17	4.71		

FEAR QUESTIONNAIRE (GLOBAL PHOBIA SCALE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	29.44	16	1.84		
SESSION	32.11	1	32.11	17.45	.001
GROUP BY SESSION	5.44	1	5.44	2.96	.105

TWO (GROUP) BY THREE (PERIOD) REPEATED MEASURES ANALYSIS OF VARIANCE ON SESSION ONE MEASURES OF TONIC PHYSIOLOGICAL ACTIVITY.

RESPIRATION RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	249.18	1	249.18	2.40	0.141
ERROR	1661.63	16	103.85		
PERIOD	2.26	2	1.13	0.24	0.785
PERIOD BY GROUP	21.81	2	10.91	2.36	0.111
ERROR	147.93	32	4.62		

THORACIC RESPIRATION DEPTH

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	173.24	1	173.24	6.30	0.023
ERROR	440.07	16	27.50		
PERIOD	7.69	2	3.85	1.87	0.169
PERIOD BY GROUP	8.44	2	4.22	2.06	0.144
ERROR	65.65	32	2.05		

ABDOMINAL RESPIRATION DEPTH

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	56.39	1	56.39	3.86	0.067
ERROR	233.67	16	14.60		
PERIOD	4.35	2	2.18	2.35	0.111
PERIOD BY GROUP	5.12	2	2.56	2.76	0.078
ERROR	29.61	32	0.92		

PEAK HEART RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	377.73	1	377.73	0.75	0.399

ERROR	8063.16	16	503.95		
PERIOD	61.68	2	30.84	4.40	0.020
PERIOD BY GROUP	12.24	2	6.12	0.87	0.427
ERROR	224.23	32	7.01		

LOW HEART RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	105.45	1	105.45	0.20	0.657
ERROR	8248.39	16	515.52		
PERIOD	54.77	2	27.38	3.85	0.032
PERIOD BY GROUP	28.57	2	14.28	2.01	0.151
ERROR	227.44	32	7.11		

NUMBER OF SPONTANEOUS SCRs

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	3.63	1	3.63	0.15	0.702
ERROR	383.18	16	23.95		
PERIOD	12.48	2	6.24	1.07	0.354
PERIOD BY GROUP	5.81	2	2.91	0.50	0.612
ERROR	186.37	32	5.82		

ONEWAY ANALYSIS OF VARIANCE BETWEEN GROUPS ON INITIAL MEASURES OF PHYSIOLOGICAL REACTIVITY.

HEART RATE ACCELERATION

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	80.22	1	80.22	1.03	.326
WITHIN GROUPS	1248.22	16	78.01		
TOTAL	1328.44	17			

HEART RATE DECELERATION

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	34.72	1	34.72	1.09	.312
WITHIN GROUPS	510.22	16	31.89		
TOTAL	544.94	17			

SKIN CONDUCTANCE RESPONSE (AMPLITUDE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	.74	1	.74	3.30	.088
WITHIN GROUPS	3.62	16	.23		
TOTAL	4.36	17			

SKIN CONDUCTANCE RESPONSE (RECOVERY TIME)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
BETWEEN GROUPS	4.11	1	4.11	5.11	.038
WITHIN GROUPS	12.87	16	.80		
TOTAL	16.98	17			

TWO (GROUP) BY TWO (SESSION) BY THREE (PERIOD) REPEATED MEASURES ANALYSIS OF VARIANCE ON MEASURES OF TONIC PHYSIOLOGICAL ACTIVITY.

RESPIRATION RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	51134.26	1	51134.26	308.43	0.00
GROUP	1696.15	1	1696.15	10.23	0.00

ERROR	2652.59	16	165.79		
SESSION	1240.33	1	1240.33	53.88	0.00
SXG	355.70	1	355.70	15.45	0.00
ERROR	368.29	16	23.02		
PERIOD	6.13	2	3.06	0.44	0.65
PXG	13.02	2	6.51	0.93	0.40
ERROR	223.85	32	6.99		
SXP	5.06	2	2.53	0.63	0.54
SXPXG	10.91	2	5.45	1.37	0.27
ERROR	127.70	32	3.99		

THORACIC RESPIRATION DEPTH

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	7036.39	1	7036.39	158.96	0.00
GROUP	212.86	1	212.86	4.81	0.04
ERROR	708.24	16	44.26		
SESSION	38.26	1	38.26	2.35	0.14
SXG	35.09	1	35.09	2.16	0.16
ERROR	260.19	16	16.26		
PERIOD	11.62	2	5.81	3.47	0.04
PXG	9.73	2	4.86	2.91	0.07
ERROR	53.53	32	1.67		
SXP	3.05	2	1.53	0.87	0.43
SXPXG	1.62	2	0.81	0.46	0.63
ERROR	55.93	32	1.75		

ANALYSIS OF COVARIANCE ON SESSION TWO THORACIC DEPTH MEASURES WITH SESSION ONE SCORES AS COVARIATES.

Source of Variation	S.S.	DF	M.S.	F	F PROB.
GROUP	1.32	1	1.32	0.04	0.840
COVARIATE	95.15	1	95.15	3.02	0.102
ERROR	471.85	15	31.46		
PERIOD	6.54	2	3.27	2.28	0.119
PERIOD BY GROUP	2.07	2	1.03	0.72	0.494
COVARIATE	0.01	1	0.01	0.01	0.926
ERROR	44.42	31	1.43		

ABDOMINAL RESPIRATION DEPTH

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	1638.55	1	1638.55	106.26	0.00
GROUP	183.33	1	183.33	11.89	0.00
ERROR	246.73	16	15.42		
SESSION	20.69	1	20.69	2.75	0.12
SXG	8.53	1	8.53	1.13	0.30
ERROR	120.57	16	7.54		
PERIOD	1.42	2	0.71	1.39	0.26
PXG	2.69	2	1.34	2.64	0.09
ERROR	16.29	32	0.51		

SXP	3.29	2	1.64	2.18	0.13
SXPXG	2.84	2	1.42	1.88	0.17
ERROR	24.16	32	0.75		

LOW HEART RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	471554.80	1	471554.80	648.91	0.00
GROUP	0.90	1	0.90	0.00	0.97
ERROR	11627.05	16	726.69		
SESSION	1960.11	1	1960.11	3.81	0.07
SXG	184.19	1	184.19	0.36	0.56
ERROR	8221.85	16	513.87		
PERIOD	8.02	2	4.01	0.73	0.49
PXG	62.12	2	31.06	5.64	0.01
ERROR	176.26	32	5.51		
SXP	58.30	2	29.15	7.15	0.00
SXPXG	1.05	2	0.53	0.13	0.88
ERROR	130.54	32	4.08		

PEAK HEART RATE

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	670017.61	1	670017.61	887.69	0.00
GROUP	329.00	1	329.00	0.44	0.52
ERROR	12076.59	16	754.79		
SESSION	1865.51	1	1865.51	5.07	0.04
SXG	87.37	1	87.37	0.24	0.63
ERROR	5888.47	16	368.03		
PERIOD	57.28	2	28.64	4.55	0.02
PXG	33.89	2	16.94	2.69	0.08
ERROR	201.59	32	6.30		
SXP	16.60	2	8.30	1.36	0.27
SXPXG	0.79	2	0.39	0.06	0.94
ERROR	195.35	32	6.10		

SPONTANEOUS SKIN CONDUCTANCE RESPONSES

Source of Variation	S.S.	DF	M.S.	F	F PROB.
MEAN	2966.26	1	2966.26	86.57	0.00
GROUP	31.15	1	31.15	0.91	0.35
ERROR	548.26	16	34.27		
SESSION	176.33	1	176.33	10.31	0.00
SXG	8.33	1	8.33	0.49	0.49
ERROR	273.67	16	17.10		
PERIOD	15.80	2	7.90	1.52	0.24
PXG	3.35	2	1.68	0.32	0.73
ERROR	166.18	32	5.19		
SXP	6.72	2	3.36	1.03	0.37

SXPXG	3.17	2	1.58	0.48	0.62
ERROR	104.78	32	3.27		

TWO (GROUP) BY TWO (SESSION) REPEATED MEASURES ANALYSIS OF VARIANCE ON MEASURES OF PHYSIOLOGICAL REACTIVITY USING SEQUENTIAL SUM OF SQUARES.

HEART RATE ACCELERATION

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	746.11	16	46.63		
SESSION	87.11	1	87.11	1.87	.191
GROUP BY SESSION	58.78	1	58.78	1.26	.278

HEART RATE DECELERATION

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	477.89	16	29.87		
SESSION	215.11	1	215.11	7.20	.016
GROUP BY SESSION	0.00	1	0.00	0.00	1.000

SKIN CONDUCTANCE RESPONSE (AMPLITUDE)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	2.16	16	.13		
SESSION	.17	1	.17	1.29	.273
GROUP BY SESSION	.01	1	.01	.05	.820

SKIN CONDUCTANCE RESPONSE (RECOVERY TIME)

Source of Variation	S.S.	DF	M.S.	F	F PROB.
WITHIN CELLS	7.84	16	7.84		
SESSION	0.11	1	0.11	0.03	0.855
GROUP BY SESSION	0.00	1	0.004	0.00	0.971

ANALYSIS OF COVARIANCE ON SESSION TWO SCR RECOVERY TIME SCORES WITH SESSION ONE SCORES AS THE COVARIATE.

Source of Variation	S.S.	DF	M.S.	F	F PROB.
COVARIATE					
SCRRT1	0.26	1	0.26	0.52	.823
MAIN EFFECTS					
GROUP	6.29	1	6.29	1.25	.280
EXPLAINED	6.55	2	3.27	0.65	.535
RESIDUAL	75.25	15	5.02		
TOTAL	81.79	17	4.81		